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MANAGEMENT OF THE INJURED KIDNEY

Chairman's Address

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Somewhat more than three years ago one of us (H. W. McK.) became dissatisfied with our management of the injured kidney, feeling that our treatment followed no preformed plan. For this reason, a study was made of all of the cases which we had seen at the Charlotte Memorial Hospital since 1940 in an attempt to ascertain whether we might not reach some conclusions which would permit the formulation of a working plan for the proper diagnosis and management in these cases.[1]

As a result of this study the following conclusions were reached: (1) we were not taking sufficient steps to diagnose the exact nature of the injury, that is, whether the kidney was merely contused or frankly ruptured; (2) we were content to follow the generally accepted mode of so-called conservative nonoperative management even in cases in which actual rupture of the kidney was demonstrated; (3) we were overlooking the numerous undesirable sequelae to injury of the kidney reported in the literature [2] and observed by us,[1] and (4) we were not insisting on adequate follow-up studies of the patients treated.

On the basis of this reevaluation of our cases, we have separated all traumatic injuries of the kidney into three groups, the classifications being governed not so much by the anatomic and pathologic type of the injury as by the method of diagnosis and management. We realize, of course, that no combination of diagnostic methods will afford an accurate diagnosis in every case and that exceptions will occur in any large series of cases. We propose herein to present this classification as we have conceived it and to describe the management of each group, using illustrative case histories. Since our adoption of this method of management we have treated a series of 12 patients; we will report 1 or more cases to illustrate each management group.

From the Urological Service of the Charlotte Memorial Hospital.

Read before the Section on Urology at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 9, 1949.

The photographic work was done by the Heineman Foundation and Mr. Rufus Meigs. Dr. G. Aubrey Hawes of the Department of Urology supplied case 7. Mr. Melvin Kester prepared the diagrams.

1. Lynch, K. M., Jr.: Management of the Injured Kidney: Preliminary Report, J. Urol. **60**: 371, 1948.

2. Colston, J. A. C., and Baker, W. W.: Late Effects of Various Types of Trauma to the Kidneys, Arch. Surg. **34**: 99 (Jan.) 1937.

CLASSIFICATION

In group 1 the patient gives a history of trauma followed by mild to severe pain in the loin and microscopic to macroscopic hematuria. Shock, flank tenderness and muscle rigidity may or may not be present. A flat plate roentgenogram of the abdomen shows clear psoas muscle and renal outlines. An excretory urogram usually shows diminished or absent function from the injured kidney during the first seventy-two hours after the injury. A retrograde pyelogram demonstrates a normal collecting system without extravasation and without undue angulation of the ureter. In our opinion the kidney has been demonstrated to be anatomically normal in this group and pathologically would be classified as contused.

In group 2 the signs and symptoms listed under group 1 are present, and in addition a mass in the loin may or may not be palpated. Shock, pain, hematuria, tenderness and rigidity may be more pronounced but are not necessarily so: A. An obliteration of the renal and/or psoas outlines, if not due to gas shadows, indicates perirenal hemorrhage and places the case in group 2. B. Extravasation of the dye from the collecting system on the excretory urogram (rare in the first seventy-two hours) or retrograde pyelogram demonstrates a rupture of the collecting system and places the case in group 2. Signs A and B may both be present but may occur singly. There may also be angulation of the ureter on the pyeloureterogram, but this is not often encountered. The cases in this group consist anatomically and pathologically of rupture either of the renal parenchyma or of perirenal vessels resulting in perirenal hematoma in which patients are in sufficiently good general condition to warrant study over a seventy-two hour period. Subcapsular hematoma is also included in this group.

In group 3 the presenting feature is shock, which is progressive despite transfusion. The urinary hemorrhage is profuse and commonly results from rupture of the vascular pedicle. A mass is generally palpable; pain, tenderness and rigidity are accentuated.

MANAGEMENT AND REPORT OF CASES

Patients in group 1 should be observed and treated nonoperatively.

CASE 1.—P. T., a Negro college student, aged 21, was admitted Oct. 23, 1948, shortly after he had received a blow to the left flank while playing football. He had noted gross hematuria after the injury, and a dull aching pain had developed in the flank. Examination revealed pronounced tenderness in the flank without muscle spasm or mass. The urine was only blood tinged. A flat plate roentgenogram showed clear renal and psoas muscle shadows. Excretory urograms showed no function from the lower pole of the injured kidney. A retro-

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grade study seventy-two hours after the injury revealed normal conditions. The urine became free of blood in twenty-four hours, and the patient was discharged four days later. A follow-up study of the kidney on March 20, 1949, five months later, revealed that the urine, phenolsulfonphthalein excretion and retrograde pyelogram were all normal. The final diagnosis was contusion of the kidney.

Fig. 1 (case 3).—Retrograde pyelogram showing filling defect in the lower portion of the pelvis caused by blood clots and irregular extravasation of dye into the parenchyma of the lower pole of the kidney. The psoas muscle and renal outlines are obliterated on this side.

CASE 2.—A. T., a sawmill worker aged 38, was admitted to the hospital because of dull flank pain and gross hematuria three days after having been struck in his right flank by a board thrown from a ripsaw. There was only moderate tenderness of the flank without muscle rigidity or a palpable mass. The flat plate roentgenogram showed clear renal and psoas muscle shadows. The excretory urogram showed poor filling on the right side. The retrograde pyelogram was normal. The hematuria cleared on the third hospital day. When the kidney was examined twelve months later, the urine, function on phenolsulfonphthalein test and retrograde pyelogram were normal. The final diagnosis was contusion of the kidney.

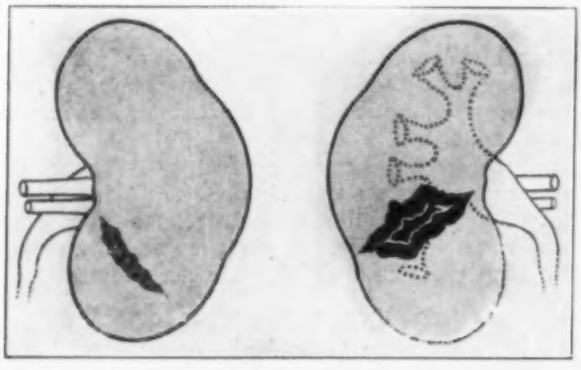

Fig. 2 (case 3).—Diagram of observations at time of operation: anterior view (left) showing superficial cortical laceration; posterior view (right) showing deep communicating rupture. This kidney was salvaged by nephrostomy drainage.

Patients in group 2 should have exploratory operations performed approximately seventy-two hours after the injury, at which time coagulation of the extravasated blood would have occurred and infarction would have become readily discernible, thereby not only facilitating the exploratory operation technically but also affording the surgeon a clearer conception of whether the kidney should be salvaged or removed, thereby minimizing the number of nephrectomies performed in this group. Salvage of the kidney may be attempted in a number of these cases by various methods, such as partial nephrectomy, ribbon gut suture over fat[3] or gelfoam®,[4] or nephrostomy drainage. We chose the McIver splinting nephrostomy tube in order to maintain the caliber and position of the ureter in face of cicatrizing scar tissue, because of the excellent drainage afforded and because of the absence of trauma on removal of the tube. We usually remove the tube seven to twelve days after the operation, believing that healing should be well advanced at this time. In our experience all wounds have healed promptly within twenty-four to forty-eight hours after removal of the tube.

Fig. 3 (case 3).—Retrograde pyelogram two months after the operation.

CASE 3.—C. S., a white man aged 38, was admitted to the hospital Sept. 21, 1947, approximately thirty minutes after a fall from a moving automobile, at which time he struck his left flank against the curb. He complained of severe pain in the left flank and appeared to be in mild shock with a blood pressure of 90 systolic and 50 diastolic. He was given ¼ grain (15 mg.) of morphine and 400 cc. of blood plasma with a resultant rise in blood pressure to 134 systolic and 80 diastolic. The left flank was rigid and exquisitely tender with no palpable mass. The urine was grossly bloody. A flat plate roentgenogram of the abdomen showed complete obliteration of the left renal and psoas muscle outlines. Excretory urograms revealed diminished function on the left with a small area of extravasation beyond the most inferior calyx. The urine gradually cleared, but the pain in the left flank persisted. On the fourth hospital day the patient was examined with a cystoscope and a catheter was passed to the left kidney, the urine from which was brown with old blood. A retrograde pyelogram showed an irregular filling defect of the lower portion of the pelvis and the inferior calices with extravasation of the dye into the

3. Lowsley, O. S., and Menning, J. H.: Treatment of Rupture of the Kidney, J. Urol. **45**: 253, 1941.

4. MacDonald, S. A., and Matthews, W. H.: Fibrin Foam and Gelfoam in Experimental Kidney Wounds, J. Urol. **57**: 802, 1947. Lynch.[1]

renal parenchyma (fig. 1). An exploratory operation on the left kidney was performed immediately after cystoscopy and ninety-six hours after the injury, with a preoperative diagnosis of rupture of the lower pole of the kidney. The lumbodorsal fascia was a dark bluish black. The perirenal fascia was distended by approximately 300 to 400 cc. of old blood clots and macerated fatty tissue. There was already considerable organization about the kidney, which was freed and delivered only with difficulty. There was a deep ragged laceration on the posterior surface of the lower pole which communicated with the pelvis and a superficial laceration on the anterior surface (fig. 2). A pyelotomy was performed, and a McIver nephrostomy tube was inserted through the ruptured area without inciting hemorrhage. The patient's postoperative course was smooth, the urine becoming practically clear the day after the operation. The nephrostomy tube was removed on the eighth postoperative day, after which the wound closed promptly. The patient was discharged ambulatory and asymptomatic on the fourteenth postoperative day, at which time the urine contained only a few pus cells per high power field. Two months and eleven months postoperatively, the kidney urine was sterile on culture and a retrograde pyelogram was normal in appearance with good cupping of the inferior calices (fig. 3). Phenolsulfonphthalein dye appeared simultaneously from both sides and in equal concentration. The patient was actively working and had no complaints. The final diagnosis was communicating rupture of the lower pole of the kidney with perirenal hematoma.

Fig. 4 (case 4).—Excretory urogram showing exceedingly poor function from the upper two thirds of the left kidney with slight but definite extravasation of dye into the parenchyma in this area. The psoas muscle and renal outlines are obliterated on this side.

We believe that if an exploratory operation had been performed on this patient on admission, a nephrectomy would have been necessary because of active hemorrhage from the area of the rupture.

CASE 4.—H. W., a white girl aged 12 years, was admitted to the hospital Feb. 19, 1948, approximately forty-eight hours after she had fallen against a stone step. Rather severe pain had developed in her left flank, and she had gross hematuria. She was ambulatory on admission to the hospital and did not appear to be in much pain. The left flank was moderately tender both anteriorly and posteriorly without muscle rigidity or palpable mass. The urine was grossly bloody. A flat plate roentgenogram of the abdomen showed complete obliteration of the renal and psoas muscle shadows on the left side. Excretory urograms revealed that little dye was excreted from the upper two thirds of the kidney but that there was an irregular dispersion of this dye beyond the normal contour of the upper pelvis and caliceal systems (fig. 4). A retrograde pyelogram was not considered necessary in this case, as rupture of the upper pole of the kidney was clearly demonstrated by the flat plate roentgenogram and excretory urogram. At operation seventy-two hours after the injury the perirenal fascia was found to be bulging, to be deep blue and to contain macerated fatty tissue infiltrated with old blood. There was a laceration at the junction of the upper one third and lower two thirds of the kidney (fig. 5). This laceration extended down almost to the hilus anteriorly and just beyond the midline posteriorly. A pyelotomy was made, and a few blood clots were evacuated from the pelvis. A McIver nephrostomy tube was inserted through the laceration in the upper pole of the kidney without

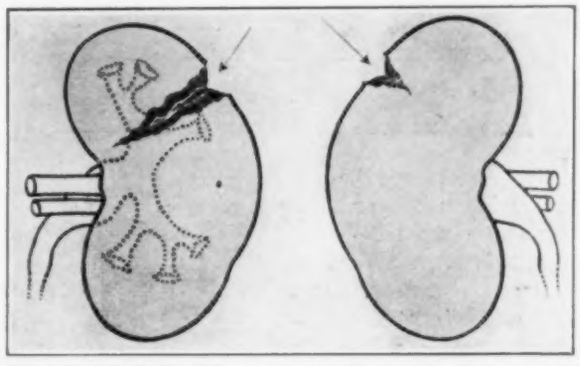

Fig. 5 (case 4).—Diagram of observations at operation. Communicating rupture is shown on anterior (left) and posterior (right) views of the kidney, which was salvaged by nephrostomy drainage.

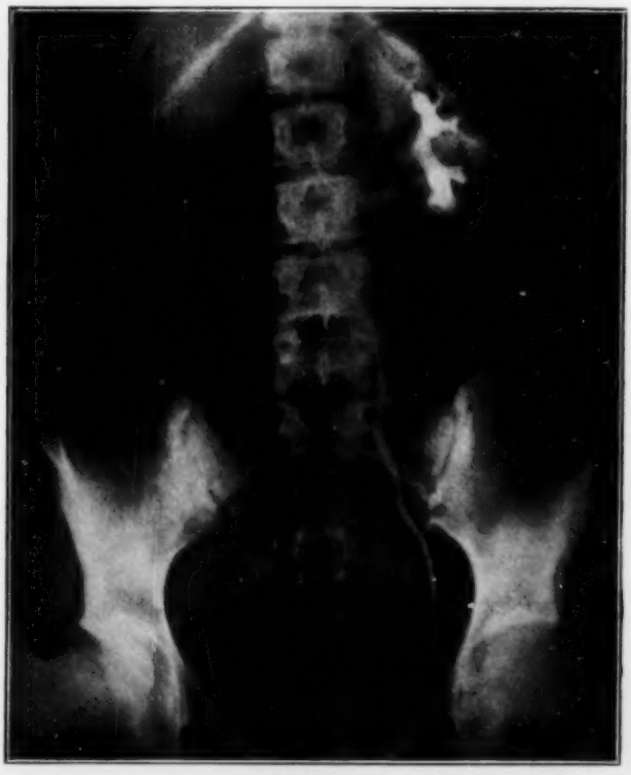

Fig. 6 (case 4).—Retrograde pyelogram thirteen months after operation. The accidental pyelotubular backflow serves to outline the indentation in the cortex at the site of the rupture.

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meeting intact tissue. The urine became grossly clear twenty-four hours after completion of the surgical procedure. The nephrostomy tube was removed on the eighth postoperative day, and the wound healed promptly. Excretory urograms which were made the day prior to discharge of the patient showed good function from the upper and middle calices of the kidney with only minimal distortion and without extravasation. On

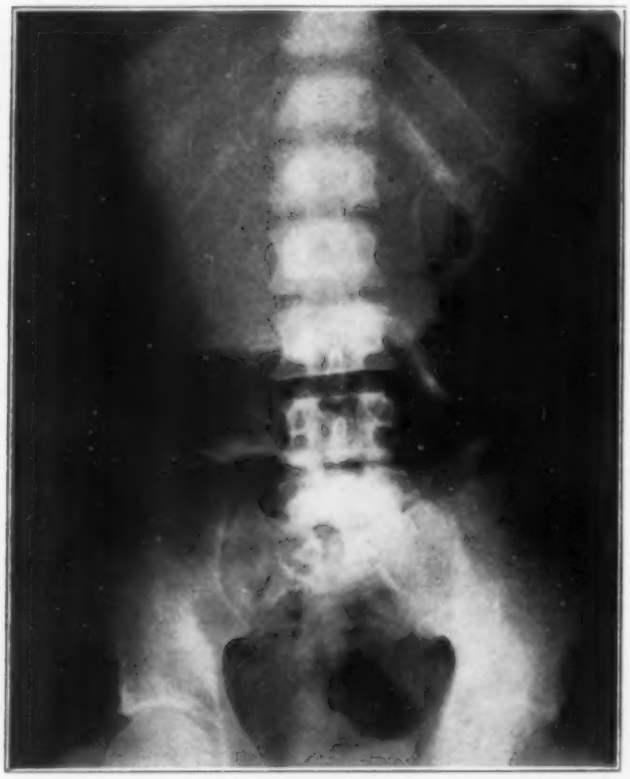

Fig. 7 (case 5).—Excretory urogram showing complete absence of function from right kidney and obliteration of renal and psoas muscle outlines.

follow-up examination thirteen months after the injury, the urine from the bladder and kidney was normal microscopically and was sterile on culture. There was an equal amount of phenolsulfonphthalein excreted from the two kidneys. A retrograde pyelogram (fig. 6) on the previously injured kidney did not reveal atrophy other than a shallow indentation at the site of the rupture. The pyelogram was normal except for accidental pyelotubular backflow. The final diagnosis was communicating rupture with perirenal hematoma.

An earlier exploratory operation on this patient probably would have necessitated nephrectomy because of active hemorrhage from the ruptured area. Non-operative treatment would have exposed the girl to possible late sequelae.

Case 5.—C. P., a Negro aged 42, was admitted to the hospital Sept. 28, 1948, approximately twenty-four hours after having been struck over his back by a chair while fighting. He had immediately experienced pain in his right flank and had subsequently noticed that his urine was bloody. On physical examination the patient was lying with his right leg flexed and was apparently in severe pain. The right flank was rigid and exquisitely tender, and there was a suggestion of an underlying mass. The urine was grossly bloody. A flat plate roentgenogram of the abdomen showed the right psoas muscle shadow and the lower half of the right renal shadow to be completely obliterated. An excretory urogram (fig. 7) showed that there was no function of the right kidney. The patient was given a cystoscopic examination forty-eight hours after admission, or seventy-two hours after his injury, and blood was seen to slowly ooze from the right urethral orifice. A right retrograde pyelogram (fig. 8) showed extravasation of the dye from the middle calices; the superior and inferior calices and pelvis were well outlined, but they appeared to be filled by a blood clot forming a cast of the pelvis. An exploratory operation on the right kidney was performed immediately, with a preoperative diagnosis of complete rupture in the middle third. The lumbodorsal fascia was dark bluish black and tense. The renal fossa was filled wtih approximately 200 cc. of old blood clots and macerated fatty tissue. There was a ragged deep laceration on the anterior and posterior surfaces of the middle third of the kidney extending down to the hilus anteriorly (fig. 9). On delivery of the kidney numerous areas of ischemic infarction were discovered. A nephrectomy was performed, from which the patient made an uneventful recovery. The final diagnosis was severe communicating rupture of the kidney with multiple ischemic infarctions and perirenal hematoma.

Earlier operation on this patient would have been technically much more difficult and dangerous because of active hemorrhage.

Case 6.—E. S., a deaf-mute student aged 18, was admitted to the hospital Nov. 17, 1947, three days after a football injury. Subsequently a dull aching pain in the flank had developed, and he had noted that his urine was bloody. Examination revealed only slight tenderness in the flank and no palpable mass. A flat plate roentgenogram disclosed poorly defined right renal and psoas muscle shadows. Excretory urograms showed poor function on the right (fig. 10). A right retrograde pyelogram showed extravasation of the dye about 1 cm. into the parenchyma from the inferior minor calyx (fig. 11). We believed that the kidney should be examined at an exploratory operation according to our postulates; however, because the flank tenderness was minimal and the urine had become clear we did not operate but discharged the patient on the sixth

Fig. 8 (case 5).—Retrograde pyelogram showing extravasation of dye from the middle calyx, shadowy pyelogram indicative of blood-clot cast filling pelvis and obliteration of renal and psoas muscle outlines.

hospital day. He was readmitted on two occasions because of gross hematuria and colicky pain, and finally an exploratory operation was performed one month after the injury. The kidney was densely adherent over the lower pole to the surrounding tissues and was only partially mobilized with the greatest of difficulty. A lower pole nephrostomy was attempted but found

to be technically impossible because of the adherence of the lower pole and pelvis to the surrounding tissues. Rather than leave the kidney and subject the patient to further bleeding, a nephrectomy was performed. The kidney (fig. 12) was normal except for the perirenal scar tissue over the lower pole and pelvis and a small area of granulation tissue in the inferior minor calyx, the point at which the rupture had been demonstrated on the initial retrograde pyelogram. The final diagnosis was pycloparenchymal rupture of the kidney with repeated secondary hemorrhage.

In this case, if we had followed our own postulates an exploratory operation would have been performed and the kidney would have been salvaged by nephrostomy drainage at the seventy-two hour period. However, by our own wishful thinking that we could treat it as a nonoperative, group 1 case we were forced to perform a nephrectomy at a late date. In reply to those who would say that the recurrent hemorrhages were due to infection carried to the ruptured area by retro-

Fig. 9 (case 5).—Kidney removed at operation seventy-two hours after the injury, showing areas of rupture and ischemic infarction.

grade pyelography, we should like to add that urine cultures were sterile on all admissions.

Patients in group 3 require immediate operative intervention as a life-saving measure as soon as shock treatment has been instituted. An excretory urogram may be obtained during this interval as supportive evidence and to evaluate the opposite kidney.

CASE 7.—J. F., a log turner aged 39, was admitted to the hospital two hours after a crushing blow to his right flank by a sawmill log-carriage. On admission the patient was in moderate shock; his bladder was distended above the umbilicus, and he was able to pass only a few blood clots via his urethra. A flat plate roentgenogram of the abdomen showed partial obliteration of the psoas muscle and renal shadows on the right side, and intravenous urography disclosed no function on the right. Attempts to evacuate the clots from the bladder by catheter revealed that the blood was coming into the bladder, probably from the ureter, as fast as it could be aspirated. Although the patient was immediately given plasma and then whole blood, he continued to progress deeper into shock. Four hours after the injury he was taken to the operating room, where a right nephrectomy was performed. There was an almost complete rupture of the kidney at the junction of the

Fig. 10 (case 6).—Excretory urogram showing poor function from right kidney. The right renal and psoas muscle shadows are poorly defined.

Fig. 11 (case 6).—Retrograde pyelogram showing extravasation of dye from most inferior calyx. The renal and psoas margins are poorly defined.

lower and upper thirds (fig. 13). The renal vein was ruptured and apparently communicated with the pelvis; this explained

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the severe bleeding down the ureter into the bladder. After removal of the kidney, the patient responded to whole blood therapy and quickly recovered from his shock. The final diagnosis was rupture of the kidney and vascular pedicle.

We believe that this patient would have bled to death if he had not been brought immediately to the

Fig. 12 (case 6).—Kidney removed at operation one month after injury, showing adherence of perinephric tissue over area of rupture demonstrated on the retrograde pyelogram.

hospital. Nephrectomy was a life-saving measure, as he was bleeding profusely from a tear in one of the major pedicle vessels.

COMMENT

Of the series of 12 cases managed according to our system of grouping, three cases were classified as group 1, 8 cases as group 2 and 1 case as group 3; in 9 cases, therefore, exploratory operations were performed. An emergency nephrectomy was mandatory according to our classification of the patient in group 3. In the series of 8 cases in group 2, 2 nephrectomies were necessary; in 3 cases of frank rupture the kidneys were salvaged by evacuation of clots and nephrostomy drainage; in 2 cases the kidneys were drained exteriorly for perirenal hemorrhage without demonstrable rupture; in 1 case the kidney was found to be grossly uninjured (this case was complicated by a fracture of the transverse process of the third lumbar vertebra on the side of the renal injury; intestinal gas prevented roentgenograms of diagnostic quality).

A comparison of this series of cases with that reported prior to our present methods of management [1] shows an increase from 30 to 70 per cent in the number of cases with exploratory operation. This is due to the fact that we formerly operated on few patients in group 2. The mortality is zero in our present series as compared to 13 per cent in our former series. We believe that this is due not only to our present method of management but also to the use of antibiotics and whole blood and to the absence of serious associated injuries in this series.

We are aware of the various contradictory modes of management of the injured kidney. Many have considered the so-called conservative nonoperative treatment preferable in the majority of cases. Sargent [5] has modified this, stating that operative intervention is not indicated in cases in which the retrograde pyelogram shows the renal pelvis to be "reasonably intact." Lowsley [3] expressed the opinion that in any case in which bleeding continues more than twenty-four hours there should be an exploratory operation; he has advocated suture of the ruptured area with ribbon gut over fat. We employed a similar method in 1 case in our previously reported series with excellent results.[1] However, in subsequent cases a nephrostomy tube has been our method of choice because of the reasons previously enumerated. In an unpublished series of experiments on dogs with injured kidneys Lowsley [6] found that without drainage the kidneys, "because contracted to a remarkable degree, rendered the dogs quite ill, and in some cases a rise in the blood pressure occurred." But when the kidney was drained this did not happen and the "damage to the kidney over a period was minimal." Colston and Baker [2] have stated that "trauma to the kidney should not be neglected. When there is doubt as to the presence of extravasation of blood or urine, prompt operation and evacuation of the clots about the kidney should be carried out, as our series of cases

Fig. 13 (case 7).—Kidney removed at emergency operation four hours after the injury, showing area of rupture. The tear in the renal vein is not clearly demonstrated in the photograph.

clearly shows that if the extravasation be allowed to remain untreated, a calcified cyst may result." Pelouze [7] has stated that "where a palliative course has been

5. Sargent, J. C.: Injuries of the Kidney, J. Urol. 53: 381, 1945.
6. Lowsley, O. S.: Personal communication to the authors, March 25, 1948.
7. Pelouze, P. S.: Office Urology, Philadelphia, W. B. Saunders Company, 1942, p. 517.

pursued with seeming success, it is well to make a study of the comparative functional values of the two kidneys a year or so after recovery, for later complete cessation of function of the injured kidney is not altogether rare." Other authors have also warned of the deleterious sequelae to injury of the kidney, such as delayed bleeding, perirenal suppuration, hydronephrosis, pyonephrosis, stone formation and atrophy.[8]

SUMMARY

A classification of renal injuries into three groups is presented. The management of each of the groups is described and representative cases reported. Patients in group 1 should be treated nonoperatively. Patients in group 2 should have exploratory operations approximately seventy-two hours after the injury, and the kidney should be salvaged if possible; a splinting nephrostomy tube is recommended. Patients in group 3 require immediate emergency nephrectomy as a life-saving measure.

A series of 12 patients treated at the Charlotte Memorial Hospital according to this system of management is analyzed.

Controversial modes of management of the injured kidney are briefly noted.

Medical Arts Building (Drs. McKay and Baird).
Charlotte Memorial Hospital (Dr. Lynch).

PULMONARY ARTERIOVENOUS FISTULA (VARIX)

Review of the Literature and Report of Two Cases

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In recent years there has developed an increasing awareness of the occurrence of pulmonary arteriovenous fistulas of congenital and often familial origin, and one now finds reports in the medical literature of lesions recognized clinically and treated surgically. Such lesions are also called angioma, cavernous hemangioma and arteriovenous varix or aneurysm. They are probably not related to malignant hemangioma of the lung, which is rare and causes widespread metastases. It appears to be probable that all benign, congenital pulmonary angiomas are of the same general nature as those observed in cases of Rendu-Osler-Weber's disease, or hereditary hemorrhagic telangiectasia, perhaps better called heredofamilial angiomatosis. Some cases merely lack the lesions of the skin and mucous membranes. The familial incidence may not be traceable in the individual case because of atavism. The hereditary nature of the disease has been discussed recently by Moyer and Ackerman.[1]

Pulmonary arteriovenous fistulas were described at necropsy by Churton[2] in 1897, Wilkens[3] in 1917, de Lange and de Vries Robles[4] in 1923, Reading[5] in 1932 and Rodes[6] in 1938. Apparently Smith and Horton[7] in 1939 made the first clinical diagnosis, and Hepburn and Dauphinee[8] in 1942 performed the first pneumonectomy for this condition. We have found reports in the literature of 41 cases, with surgical treatment employed in 23. To these we are adding 2, with surgical treatment in 1. We know of several other cases which have not been reported. Two such cases of Dr. Brian Blades are included in the tabular data of this review.

These pulmonary arteriovenous fistulas are composed of a distended, thin-walled afferent artery, distended efferent veins and an intervening loculated vascular sac or labyrinth of distended vessels. All these vascular components gradually become more dilated, and degenerative changes may occur, causing more intercommunications to form and occasionally leading to rupture with hemorrhage into the pulmonary parenchyma or pleural sac. Multiple lesions occur in more than half of the cases. The cavities are usually lined by mesothelial cells on a thin layer of connective tissue. The pathogenesis is probably "a gross deviation of capillary formation."[1] The small cutaneous and mucocutaneous telangiectases or angiomas "are characterized by disseminated abnormalities of capillaries, small venules and small arterioles. . . . They vary from typical spider nevi to pea-size hemangiomata."[1] They may involve any organ. The vessels may rupture easily. Bean[9] has differentiated between acquired spider nevi and the angiomatous lesions of hereditary hemorrhagic telangiectasia. Some patients are photosensitive, and exposure to sun rays causes hemorrhage of cutaneous lesions; however, in some cases the hemorrhagic tendency is absent.

TABLE 1.—*Pulmonary Arteriovenous Fistula Symptoms in 45 Cases*

					Not Stated
Age Diagnosed	Youngest 2 Days Average 28 Yr.		Oldest 56 Yr.		1
Sex	Male	31	Female	12	2
Color	White	43	Negro	2	
Family history	Positive	12	Negative	8	25
Age of onset of symptoms	At birth	9	Childhood	11	
	Over 12 yr.	18			7

Symptoms	Present	Absent	Not Stated
Cyanosis	33	3	9
Clubbing of fingers	31	3	11
Bruit (extracardiac)	26	3	16
Dyspnea	23	2	20
Epistaxis	14	2	29
Weakness	13	3	29
Cardiac abnormalities	9	24	12
Convulsions	9	4	32
Hemoptysis	8	2	35
Chest pain	8	3	34
Unconsciousness	8	3	34
Dizziness	7	4	34
Cerebrovascular accidents	4	..	41
Numbness	5	..	40
Splenomegaly	3	4	38
Vomiting	3	4	38
Faintness	3	3	39

8. Lynch.[1] Colston and Baker.[2] Lowsley and Menning.[3]
From the Yater Clinic and the private practice of one of us (J.F.).

1. Moyer, J. H., and Ackerman, A. J.: Hereditary Hemorrhagic Telangiectases Associated with Pulmonary Arteriovenous Fistula in Two Members of a Family, Ann. Int. Med. **29**: 775, 1948.
2. Churton: Multiple Aneurysm of Pulmonary Artery, Brit. M. J. **1**: 1223, 1897.
3. Wilkens, G. D.: Ein Fall von multiplen Pulmonalis Aneurysmen, Beitr. z. Klin. d. Tuberk. **38**: 1, 1917.
4. de Lange, C., and de Vries Robles, S. B.: Ueber Lungenangioma bei einem Säugling, Ztschr. f. Kinderk. **34**: 304, 1922-1923.
5. Reading, B.: A Case of Congenital Telangiectasis of the Lung Complicated by Brain Abscess, Texas State J. Med. **23**: 462, 1932.
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7. Smith, H. L., and Horton, B. T.: Arteriovenous Fistula of the Lung Associated with Polycythemia Vera: Report of a Case in Which the Diagnosis Was Made Clinically, Am. Heart J. **18**: 589, 1939.
8. Hepburn, J., and Dauphinee, J. A.: Successful Removal of Hemangioma of the Lung Followed by the Disappearance of Polycythemia, Am. J. M. Sc. **204**: 681, 1942.
9. Bean, W. B.: The Cutaneous Spider: A Survey, Medicine **24**: 243, 1945.

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Rendu [10] in 1896 was the first to report a case of hereditary hemorrhagic telangiectasia, and Osler [11] in 1901 reported 3 cases of the disease associated with epistaxis. Later Weber [12] reported other cases and familial groups and elaborated on the disease. None of these authors described pulmonary lesions. Since then more than 1,000 cases have been reported with lesions involving practically any area in the body.[1]

The circulatory fault resulting from pulmonary arteriovenous fistula is dependent on the amount of unoxygenated blood passing through the fistula. The cardinal symptoms are due to the chronic anoxemia produced by the incomplete aeration of blood in the lungs, and the presence and degree of symptoms therefore depends on the size and duration of the shunt. With large fistulas the bone marrow is stimulated to form more erythrocytes. The blood volume is thereby increased, most of the increment being red blood cells. Consequently both the hemoglobin and red blood cell count are increased as well as the hematocrit. Cyanosis and clubbing of the digits are other results. Occasionally hypertrophic pulmonary osteoarthropathy is noted. Other manifestations are dyspnea on exertion, hemoptysis, weakness, precordial pain, convulsions, unconsciousness, dizziness, diplopia, cerebrovascular accidents, numbness, faintness, palpitation, headache, thick speech and emesis. Many of these symptoms are due to the compensatory polycythemia. Table 1 gives the salient clinical features in the 45 cases.

A murmur is often heard over the lesion. It is usually continuous with systolic accentuation and most intense on deep inspiration, often fading on expiration. Occasionally the murmur is entirely diastolic and increased on inspiration. The heart is usually not enlarged, because the strain on the right ventricle is slight because of low blood pressure in the pulmonary circulation. The cardiac output remains normal. The blood pressure is usually normal. The electrocardiogram is also usually normal, but occasionally there is right or left axis deviation. Nodal tachycardia and premature beats have been noted during operative intervention. The venous pressure is usually within normal limits. The arm-to-lung time and arm-to-tongue time are usually within normal limits. The arterial oxygen saturation is decreased, sometimes to as low as 63 per cent, and decreases still more on exercise (in 1 case to 59 per cent). The vital capacity may be decreased. Biochemical examinations give normal results, but the carbon dioxide–combining power of the blood or alkali reserve may be slightly depressed. The spleen is occasionally enlarged. After operation with removal of the only or main fistula the foregoing alterations promptly disappear, the clubbing of the digits disappearing less rapidly. Table 2 gives the laboratory data in the 45 cases.

The radiologic appearance of the lesion is characteristic. Even in plain roentgenograms the diagnosis may be made, but angiocardiograms are pathognomonic. On the routine roentgenogram a lobulated opacity is seen, with bandlike linear or sinuous opacities connected with the hilus. These are sharply shown in angiocardiograms, the bandlike connecting densities denoting the afferent and efferent pulmonary vessels. Fluoroscopic examination occasionally shows pulsations in the lobulated density and/or the hilus. The Valsalva test (deep inspiration followed by forcible expiration against the closed glottis) may result in some decrease in the size of the shadow, whereas Mueller's test (deep expiration followed by forcible inspiration against the closed glottis) may cause an increase. Roentgenograms are best made during the Valsalva test. The pulsation may be proved by kymographic studies.

The differential diagnosis is not difficult. Polycythemia rubra vera is eliminated by the facts that the patient is usually older, has immature leukocytes and basophilia and lacks the typical pulmonary shadow. Splenomegaly may occur in both conditions but is commoner in polycythemia vera. In rare cases, however, there is a rounded, homogeneous pulmonary shadow, perhaps an infarct, in polycythemia vera, as described by Hirsh,[13] but the lesion is not lobulated and does not have a linear band connected with the hilus. Congenital heart disease is excluded by the absence of cardiac

TABLE 2.—*Main Laboratory Data in 45 Cases of Pulmonary Arteriovenous Fistula*

Roentgenogram Positive	Regular 41	Angiography 10	Kymography 2	Planography 2	
		Present		Absent	Not Stated
Polycythemia		28		5	12
Oxygen unsaturation (below 90%)		12 (lowest 63%)		.....	33
R. B. C. (5,000,000)		Above.....28 (highest 11,450,000)		Below.... 4	13
Hgb. (100%, 15 Gm.)		Above.....25 (highest 24.9 Gm., 155%)		Below.... 5	15
Hematocrit (50 mm.)		Above.....10 (highest 82)		Below.... 3	32
Blood volume		Increased.... 9 (highest 12,900 cc., 161 cc./Kg.)		.....	36

TABLE 3.—*Pulmonary Arteriovenous Fistula*

Number and Location of Lesions, Treatment and Outcome in 45 Cases							
Number of lesions	Solitary.....18	Multiple.....27	Bilateral.....13				
Location of lesions	Lower lobe.....29	Middle lobe.....14	Upper lobe.....10	Not stated..... 8			
Surgical treatment (total 26)	Lobectomy.....19	Pneumonectomy..... 5	Ligation only..... 1	Excision only.. 1			
Outcome—not operated	Died of the condition.... 6	Died of other conditions 3	Unchanged..... 2	Not stated..... 8			
operated	Cured.....17	Improved..... 2	Died postoperatively.... 2	Not stated..... 4	Unchanged... 1		

10. Rendu: Epistaxis repetée chez un sujet porteur de petits angiomes cutanés et muqueaux, Bull. et mém. Soc. méd. d. hôp. de Paris **13**: 731, 1896.

11. Osler, W.: A Family Form of Recurring Epistaxis Associated with Multiple Telangiectases of the Skin and Mucous Membranes, Bull. Johns Hopkins Hosp. **11**: 333, 1901.

12. Weber, F. P.: Multiple Hereditary Developmental Angiomata (Telangiectases) of the Skin and Mucous Membranes Associated with Recurring Hemorrhages, Lancet **2**: 160, 1907; Developmental Telangiectatic Hemorrhages and So-Called "Telangiectasia," Familial and Non-Familial, Brit. J. Child. Dis. **21**: 198, 1924; Hemorrhagic Telangiectasia of the Osler Type—"Telangiectatic Dysplasia," Brit. J. Dermat. **68**: 182, 1936.

13. Hirsh, I. S.: Pulmonary Changes in Polycythemia Vera, Radiology **26**: 469, 1936.

observations of such a lesion in cases of pulmonary arteriovenous fistula and the absence of the typical roentgenographic lesion in congenital heart disease. Infrequently, aneurysm of the pulmonary artery needs to be considered.[14] In some cases there are cyanosis, clubbing and polycythemia; dyspnea, cough and hemoptysis are not uncommon. The roentgenographic shadow of pulmonary aneurysm is connected with the mediastinum and usually pulsates; angiocardiography is most important in revealing its nature. The roentgenographic lesion of pulmonary varix is apt to be confused with an inflammatory or neoplastic lesion, but roentgenologists are becoming cognizant of the differentiation and are suggesting the proper diagnosis. For internists the diagnosis is most difficult before the appearance of polycythemia and cyanosis.

In the 45 cases listed the diagnosis was finally established clinically in 36, at autopsy in 8 and at operation in 1. Prior diagnoses were polycythemia rubra vera in 7, congenital heart disease in 6, tuberculosis in 5, pneumonia in 3, pulmonary tumor in 2 and 1 each of Hodgkin's disease, intercostal aneurysm, renal disease, arthritis, "laziness" and "hypotension." In 22 cases a prior diagnosis was not reported.

Pulmonary fistula differs in its physiologic effects from peripheral fistula in that with the latter the increased blood volume is the result of a fairly proportional increase in plasma and erythrocytes, the heart may be enlarged, the oxyhemoglobin saturation is normal, the cardiac output is increased and cyanosis and clubbing are not present.

Treatment consists in lobectomy or partial resection for solitary lesions, or even pneumonectomy if there are multiple fistulas in all lobes of one lung (table 3).

Pneumothorax is of no value. Patients with small fistulas that do not produce the alterations of large fistulas may be kept under observation and operated on if such alterations develop. However, pulmonary hemorrhage is an ever present threat and may be fatal. The increased likelihood of thrombosis due to the polycythemia must be kept in mind at the time of operation. If phlebothrombosis occurs in a peripheral vein postoperatively, venous ligation and/or anticoagulant therapy should be used. Phlebotomy with withdrawal of blood and replacement with plasma may be advisable preoperatively.

The prognosis with surgical treatment is good, but the presence of multiple fistulas may cause trouble years later if the smaller fistulas cannot be removed along with the most important lesions. Among the serious complications, besides pulmonary hemorrhage, thrombosis due to polycythemia is important, particularly in the cerebral vessels. Table 4 gives the incidence of cerebrovascular complications in the 45 cases.

TABLE 4.—*Pulmonary Arteriovenous Fistula*

	Cerebral Manifestations in 45 Cases					
Case*	Paralysis or Weakness of Extremities	Numbness or Paresthesia	Dizziness or Vertigo	Unconsciousness	Convulsions	Other
3	.....	.....	..	..	+	
4	Left arm and leg	.....	..	..	..	
6	.....	.....	..	..	+	
7	.....	Hands and feet	+	..	..	Tinnitus; diplopia
8	.....	.....	+	..	..	
13	.....	.....	+	+	..	
15	.....	Left arm	+	..	+	
18	Left arm and leg	.....	..	+	+	
20	.....	Left side	+	+	..	
21	.....	.....	..	+	..	Headaches; blurred vision
22	.....	.....	..	..	Left side	
23	.....	.....	+	..	..	Tinnitus
29	Left arm and leg	.....	..	+	+	
30	.....	.....	..	..	+	EEG epileptic curve
37	.....	.....	+	..	..	Blurred vision; diplopia
40	Left hand	.....	..	+	+	Brain abscess
41	Right hemiplegia	.....	..	+	Left side	Aphasia
43	.....	.....	..	+	..	Mental confusion
44	Right side	+	..	..	..	Aphasia
45	.....	+	..	..	..	
Total 20	6	5	7	8	9	9

* The cases are numbered as in table 5; cases 44 and 45 are cases 1 and 2, respectively, of this article.

REPORT OF CASES

CASE 1 (case 44 in tables 4 and 5).—Mrs. E. R., a white housewife aged 42, had been under the care of one of us (J. F.) for a year and a half. She had had recurrent fleeting attacks of numbness and tingling of the limbs, pain in the left side of the chest without relation to exertion, dyspnea on exertion and cyanosis. Her childhood had been normal, but cyanosis had been noted since she was 10 years of age. The dyspnea had been of recent origin. She had had chickenpox, measles, pertussis, pneumonia (at 10), phlebitis at 25, at childbirth, and an appendectomy and removal of an ovarian cyst at 22 years of age. She had had six pregnancies with four living children. In 1938 she had taken one of her children to a large clinic because of a possible congenital cardiac defect. At that time she, too, had been examined. A systolic humming murmur had been heard low in the interscapular region, louder when she held her breath. A consolidation had been noted in the lower lobe of the right lung. A diagnosis of Eisenmenger's complex had been suggested. An electrocardiogram showed a sinus rhythm with a P-R interval of 0.12 to 0.16 second. In 1940 the patient had had a "stroke" involving the right side of the face and the right arm with some disturbance of speech, all clearing up in three days. In 1945 she had had another "stroke" manifested by aphasia of less than a day's duration. The phlebitis previously noted had occurred with the birth of the son already referred to, who was said to have an interauricular septal defect; since then she had had recurrent pain in the legs, for which venous ligation had recently been performed. On first examination the cyanosis was noted as well as clubbing of the fingers. The second pulmonic sound was accentuated. A roentgenogram of the chest on May 25, 1946 showed a lesion in the lower lobe of the right lung. The hemoglobin level was 114 per cent; the red blood cells numbered 5,760,000 and the white blood cells 6,600 with a normal differential. The urine was normal and reaction to a Wassermann test was negative. On June 26, 1946 a humming murmur was heard medial to the angle of the right scapula. In July 1946 the hemoglobin was 114 per cent (18 Gm.) and the red blood cells numbered 7,400,000 with the cell pack 45 per cent. The total plasma volume was 4,000 cc. and the whole blood volume 7,272 cc., or 114 cc. per kilogram of body weight. Because of the polycythemia phlebotomies were performed. The blood pressure ranged from 110 systolic and 70 diastolic to 135 systolic and 100 diastolic. Again, on Nov. 29, 1947, a continuous humming murmur was noted in the right side of the chest posteriorly below the scapula, accentuated by deep inspiration. At that time the diagnosis of congenital arteriovenous fistula was made (by J. F.). The venous pressure was 8 cm., and the vital capacity was 93 per cent of normal.

14. Deterling, R. A., and Clagett, O. T.: Aneurysm of the Pulmonary Artery: Review of the Literature and Report of a Case, Am. Heart J. 34: 471, 1947.

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When first examined at the Yater Clinic on Dec. 4, 1947, the patient complained mainly of dyspnea on exertion. She was well developed and nourished. Her blood pressure was 130 systolic and 70 diastolic, her pulse rate was 86. She was moderately cyanotic and had slight clubbing of the fingers. The heart was not enlarged on physical examination, fluoroscopic examination or in roentgenograms. On deep inspiration with the breath held, a long, high-pitched systolic murmur was heard below the right scapula in an area corresponding to the upper half of the lower lobe of the right lung. It was heard down into the lower part of the right axilla and below the right breast. It was continuous, with slight systolic and diastolic accentuation. A roentgenogram disclosed opacities in the right lower lung field (figs. 1 and 2). The lateral view revealed a lesion which in appearance suggested a bunch of grapes. Angiocardiograms made at the Cardiovascular Research Unit of the Veterans Administration Hospital, Washington, D. C., showed a large angiomatous lesion in the lower lobe of the right lung with large vessels entering and leaving it (figs. 3 and 4). An electrocardiogram showed low voltage in the standard leads with normal voltage of the QRS complexes in the precordial leads, indicating that the main electromotive force was directed anteroposteriorly rather than in the plane of the limb leads. Lobectomy was advised and was performed with the patient under thiopental sodium (pentothal®) and ethylene anesthesia with 240 units of d-tubocurarine chloride by one of us (H. M. G.) on Feb. 13, 1948, as follows:

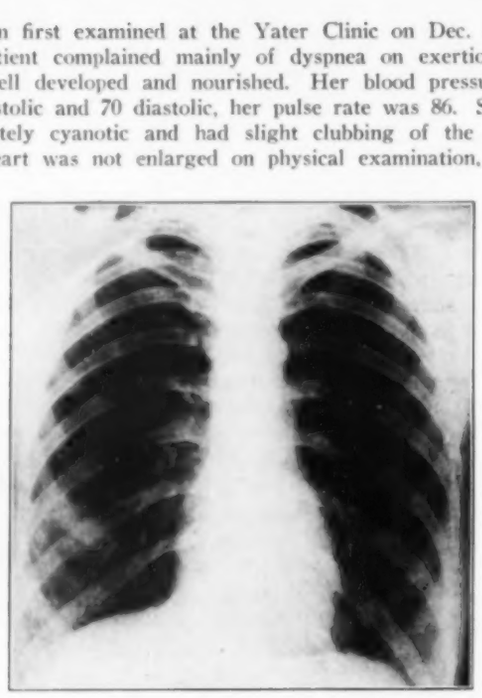

Fig. 1 (case 1).—Roentgenogram showing irregular opacity in lower right lung field.

Fig. 2 (case 1).—Roentgenogram showing in lateral view "bunch of grapes" opacity typical of pulmonary varix.

With the patient in the left lateral decubitus the chest was opened through the bed of the seventh rib. When the pleural space was entered, the lower lobe of the right lung was found to be free of all adhesions. On its lateral surface was a circular area approximately 5 by 5 cm. which obviously represented the peripheral portion of the arteriovenous communication. There was a palpable thrill over the entire lower lobe, but it was maximal at this point. Examination of the interlobar fissure revealed an unusually enlarged pulmonary artery with a palpable thrill along its course. It was decided that a right lower lobectomy would be required, the fistula being intimately incorporated within the lobe. The pulmonary ligament was first severed between clamps and ligated. The pleura was then dissected from the hilus posteriorly as well as in the interlobar fissure, and an individual dissection and ligation was done, first tying off the pulmonary artery in the anterior-superior position and then the pulmonary veins inferiorly. Both the artery and vein were unusually large, probably because of the presence of the aneurysm, and were extremely friable. However, it was possible to place double ligatures of no. 1 braided black silk followed by a transfixion suture of the same material in each instance. Closure of the lower lobe bronchus just below the origin of that of the middle lobe was accomplished with no. 0 black silk mattress sutures beneath the clamp followed by a running chromic no. 1 surgical gut over-and-over suture. There was no unusual bleeding at any time. The patient's condition was satisfactory throughout the operation. Prior to closure the pleural space was irrigated with warm saline solution, and no leakage of the bronchus was found. A no. 26 catheter was inserted through the ninth interspace in the posterior axillary line with gravity bottle drainage. The chest was closed in the usual manner while simultaneous positive pressure was administered and suction was applied to the intercostal catheter. At the end of the operation the patient's color was good, and the blood pressure was maintained after returning the patient to dorsal decubitus.

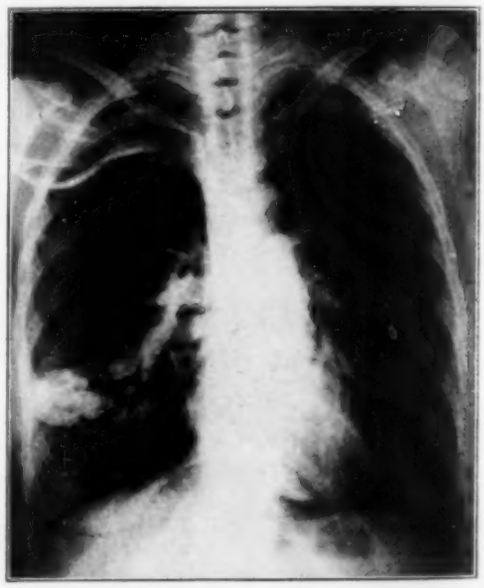

Fig. 3 (case 1).—Angiocardiogram (two seconds), showing typical appearance of pulmonary varix.

Hematologic, circulatory and blood chemical examinations were made before and after operation. The highest preoperative erythrocyte count was 7,400,000 with 114 per cent hemoglobin and a hemotocrit reading of 55 per cent. Two days before operation the erythrocyte count was 4,400,000 with 88 per cent hemoglobin. Three months after operation the erythrocyte count

was 3,750,000 with 74 per cent hemoglobin and a hematocrit reading of 39 per cent. The highest preoperative blood volume was 7,272 cc. Two days before operation it was 6,843 cc., of which 3,760 cc. was plasma. Eight days after operation the blood volume was 6,409 cc., of which 4,320 cc. was plasma; four months later the blood volume was 6,688 cc., of which 4,080 cc. was plasma. One month before operation the arm-to-tongue circulation time (calcium gluconate) was 8.5 seconds and the arm-to-lung time (ether) 4.2 seconds. These times were essentially the same after operation. The serum calcium determination was 8 mg. per hundred cubic centimeters before and 10.5 mg. after operation. The carbon dioxide–combining power was 65.5 volumes per cent before and 69 volumes after operation. Serum sodium determination was 344 mg. per hundred cubic centimeters before and 374 mg. after operation. Serum potassium was 19 mg. per hundred cubic centimeters before and 15 mg. after operation. Oxygen determinations of venous and arterial blood were made, but because the blood clotted in some specimens these values are not reported.

Fig. 4 (case 1).—Angiocardiogram (three seconds), lateral view.

The heart rate ranged from 80 to 96 before operation and between 72 and 76 per minute afterward.

The postoperative course was uneventful. The patient was out of bed and walking after a week. The dyspnea and cyanosis no longer existed. The clubbing of the fingers was not evident after six months. There was no deformity of the chest. Fluoroscopic examination revealed normal conditions except for slight elevation of the right hemidiaphragm.

Examination of the excised lobe revealed a large cluster of exceedingly thin-walled, mesothelium-lined blood vessels of various sizes, the largest more than 2 cm. in diameter (figs. 5 and 6) lying in the parenchyma of the lung just beneath the pleura laterally. Externally this "angioma" felt soft. Several large vessels led to and away from the cluster. The vascular channels of the cluster apparently intercommunicated freely, but they could not be differentiated into arteries and veins.

CASE 2 (case 45 in tables 4 and 5).—Mr. C. B., a farmer aged 42, came in for examination Aug. 12, 1948 because of pains in the joints. For three to four years he had been having attacks about once a week of severe pain, swelling, redness and heat in various large joints, usually one at a time, lasting for about twenty-four hours. In the interval he was entirely well. These attacks were preceded by numbness of the hands and arms during the night and were accompanied with stiffness of the adjoining muscles. There had been an occasional mild epistaxis for twelve years, and for six or seven years he had

Fig. 5 (case 1).—Gross specimen of removed lower lobe of right lung cut through main part of varix.

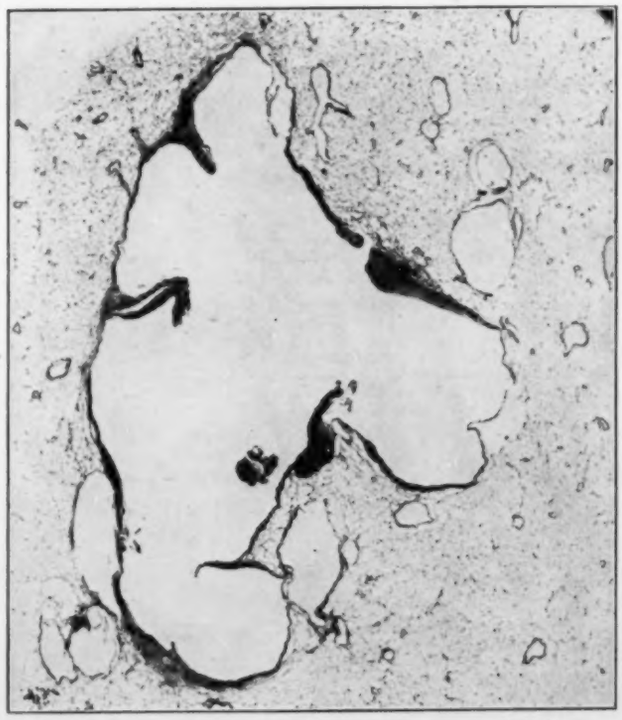

Fig. 6 (case 1).—Section through portion of varix (× 6).

been aware of small "blood blisters" on the lower lip which bled easily. He had worked hard as a farmer without dyspnea, was married and had five healthy children. He was illiterate

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TABLE 5.—*Data in 45 Cases of*

Case	Author	Age	Sex	Family History	Onset of Symptoms	Cyanosis	Clubbing	Dyspnea	Epistaxis	Hemangiomas	Hemoptysis	Weakness	Chest Pain	Convulsions	Unconsciousness
1	Churton,[2] 1897	12	M	..	11 yr.	..	..	..	+	..	+	+	..	..	..
2	Wilkens,[3] 1917	23	F	..	17 yr.	+	+	..	..	..	..	..	..	..	..
3	de Lange and de Vries Robles,[4] 1923	10 wk.	..	..	Infancy	..	..	+	..	..	..	..	..	+	..
4	Reading,[5] 1932	4	F	..	Birth	+	+	..	..	..	..	+	..	..	..
5	Bowers, W. F.: Nebraska M. J. **21**: 55, 1936	2 days	M	..	2d day	..	..	+	..	..	..	..	..	..	..
6	Rodes,[6] 1938	25	M	—	Birth	+	+	..	..	+	..	..	+	+	..
7	Smith and Horton,[7] 1939	40	M	—	Birth	+	+	+	..	..	..	..	+	..	..
8	Hepburn and Dauphinee,[8] 1942	23	F	..	15 yr.	+	+	+	..	..	..	..	..	..	..
9	Goldman, A.: Dis. of Chest. **9**: 479, 1943	22	M	—	Infancy	+	+	+	..	+	..	..	+	..	..
10	Adams, W. E.; Thornton, T. F., Jr., and Eichelberger, L.: Arch. Surg. **49**: 51, 1944	24	M	+	6 yr.	+	+	..	+	+	..	..	..	..	..
11	Janes, R. M.: Brit. J. Surg. **31**: 270, 1943-1944	30	M	..	.....	+	..	..	..	+	+	..	..	..	..
12	Jones and Thompson, W. P.: J. Thoracic Surg. **13**: 357, 1944	24	F	..	Birth	+	+	..	..	..	..	..	..	..	..
13	Alexander, W. S.: New Zealand M. J. **44**: 180, 1945	41	M	..	For years	+	+	+	..	..	..	..	+	..	+
14	Rundles, R. W.: Am. J. M. Sc. **210**: 76, 1945	56	M	..	14 yr.	..	..	+	+	+	..	+	+	..	..
15	Sisson, J. H.; Murphy, G. E., and Newman, E. V.: Bull. Johns Hopkins Hosp. **76**: 93, 1945	45	F	—	22 yr.	+	+	+	..	..	+	..	..	..	..
16	Makler, P. T., and Zion, D.: Am. J. M. Sc. **211**: 261, 1946	21	M	—	14 yr.	+	+	+	+	+	..	..	..	..	..
17	Lindgren, E.: Acta radiol. **27**: 585, 1946	30	M	..	14 yr.	+	+	+	..	..	..	+	..	..	..
18	Lindgren, E.: Acta radiol **27**: 585, 1946	29	M	+	Childhood	+	+	+	..	+	+	+	..	..	+
19	Lindgren, E.: Acta radiol. **27**: 585, 1946	25	F	..	.....	+	+	+	..	..	+	+	..	..	..
20	Beierwaltes, W. H., and Byron, F. X.: J. A. M. A. **134**: 1069, 1947	27	F	+	Birth	+	+	+	+	+	..	..	..	..	+
21	Bisgard, J. D.: Ann. Surg. **126**: 965, 1947	29	M	..	21 yr.	+	+	+	..	+	..	+	+	..	+
22	Boerema, I., and Brilman, R. P.: Nederl. tijdschr. v. geneesk. **91**: 2736, 1947	10	M	..	Birth	+	+	+	..	..	+	..	..	..	..
23	Burchell, H. B., and Clagett, O. T.: Am. Heart J. **34**: 151, 1947	20	M	..	8 yr.	+	+	..	..	..	..	..	..	..	..
24	Goldman, A.: J. Lab. & Clin. Med. **32**: 330, 1947	32	M	..	.....	+	+	..	..	..	..	..	..	..	..
25	Mallory, T. B.; Castleman, B., and Parris, E. E.: New England J. Med. **236**: 951, 1947	23	F	..	Childhood	+	+	..	..	..	..	+	+	..	..
26	Sweet, R. H., in discussion on Mallory, Castleman and Parris; personal communication to author	2	M	..	Birth	+	+	..	..	..	..	..	..	..	..
27	Salvesen, H. A.; Tidsskr. f. Den Norske Laegefor. **67**: 460, 1947	25	F	..	Childhood	+	+	—	..	..	..	..	..	..	..
28	Watson, W. L.: Surgery **22**: 919, 1947	27	M	..	25 yr.	..	..	..	..	..	..	..	..	..	..
29	Watson, W. L.: Surgery **22**: 919, 1947	21	M	+	19 yr.	+	..	+	..	+	..	..	..	+	+
30	Williams, C. F., and Flink, E. B.: J. Lab. & Clin. Med. **32**: 1401, 1947	33	M	+	30 yr.	..	..	..	+	+	..	..	..	+	..
31	Whitaker, W.: Thorax **2**: 58, 1947	44	F	+	Birth	+	+	+	+	+	..	..	..	..	..
32	Whitaker, W.: Thorax **2**: 58, 1947	33	M	..	.....	—	—	..	+	+	..	..	..	..	..
33	Cleland, W. P.: Thorax **3**: 48, 1948	..	..	..	.....	+	+	+	..	+	+	..	..	..	..
34	Maier, H. C.; Himmelstein, A.; Riley, R. L., and Bunin, J. J.: J. Thoracic Surg. **17**: 13, 1948	20	F	..	Birth	+	+	+	..	..	..	+	..	..	..
35	Swanson, P. C.: discussion on Maier, Himmelstein, Riley and Bunin	25	M	..	.....	..	—	..	..	..	..	..	..	..	..
36	Moyer, J. H., and Ackerman, A. J.,[1] 1948	..	M	+	7 yr. before	..	..	..	+	+	..	..	..	..	..
37	Moyer, J. H., and Ackerman, A. J.,[1] 1948	26	M	+	21 yr.	+	+	..	+	+	..	+	..	..	..
38	Moyer, J. H., and Ackerman, A. J.,[1] 1948	28	M	+	23 yr.	+	..	..	+	+	..	+	..	..	..
39	Wodehouse, G. E.; Himmelstein, A.; Riley, R. L., and Bunin, J. J.: J. Thoracic Surg. **17**: 408, 1948	21	M	..	.....	..	..	..	..	..	..	..	..	..	..
40	Wodehouse, G. E.; Himmelstein, A.; Riley, R. L., and Bunin, J. J.: J. Thoracic Surg. **17**: 408, 1948	13	M	..	5 yr.	+	+	+	..	..	..	+	..	+	+
41	Baer, S.; Behrend, A., and Goldburgh, H. L.: Am. Heart J., to be published	21	M	+	13 yr.	+	+	..	—	..	+	..	..	+	+
42	Blades, B.: Personal communication to the authors	20	M	—	None	—	—	—	—	—	—	—	—	—	—
43	Blades, B.: Personal communication to the authors	22	M	+	21 yr.	+	+	+	+	+	—	+	—	—	+
44	Yater, Finnegan and Giffin, 1949	42	F	—	10 yr.	+	—	—	—	—	—	—	+	—	—
45	Yater, Finnegan and Giffin, 1949	42	M	+	30 yr.	—	—	—	—	—	—	—	—	—	—

Pulmonary Arteriovenous Fistula

Dizziness	Cerebrovascular Accidents	Numbness	Faintness	Bruit	Splenic Enlargement	Hemaglobin %	Hemaglobin Gm.	Erythrocytes, Millions	Hematocrit	Arterial Oxygen Saturation, %	Blood Volume, Cc.	Location and Number of Lesions	Treatment	Course
..	..	..	..	+	..	...	....	....	..	....	.....	Bilat. 7	0	Autopsy
..	..	..	..	+	..	...	....	....	..	....	.....	Mult. R.M., R.L., L.L.	0	Death by hemothorax
..	..	..	..	..	..	...	....	....	..	....	.....	Mult. R.U. 3, L.U. 2	0	Death
..	..	..	..	+	..	102	....	6.70	..	....	.....	Mult. L. lung	0	Death by brain abscess
..	..	..	..	..	..	...	....	....	..	....	.....	Mult. R.L. & L.L.	0	Death by hemothorax
..	..	..	..	..	..	118	....	7.54	..	....	.....	R.M., R.L., L.U. 3	0	Death by hemoptysis
+	..	+	..	+	..	...	23.7	6.47	71	....	6,068; 121/Kg.	R.L.L.	0	
+	..	..	+	—	..	146	22.8	9.60	80	73.0	8,500	R.M., R.L.	Pneumonectomy	Cured
..	..	..	..	..	+	137	....	11.45	..	70.0	8,170	L. midlung	Pneumonectomy	
..	..	..	..	—	..	...	23.0	7.20	..	71.0	.....	L.L.L. 1, L.U. 2	Pneumonectomy	Cured
..		..	..	+	..	...	....	....	..	....	.....	R.M., R.L., L.L.4	Local excision	Improved
..	..	..	..	+	..	130	....	7.50	..	....	.....	Mult. R. lung	Pneumonectomy	Cured
+	..	..	..	+	..	...	20.4	8.50	..	....	.....	Mult. bilat.	0	Death by coronary
..	..	..	..	+	+	26	....	3.10	..	....	.....	R.M.L. 1	Transf. only	
+		+	..	+	..	...	14.5	5.87	..	....	.....	Mult. L.L., R.M.	0	Death by iodine reaction ("diodrast")?
..		..	..	..	..	...	19.5	7.70	55	....	.....	Mult. R. 3, L. 1	0	
..		..	..	+	..	55	....	4.30	..	89.9	.....	L.L.L. 1	0	
..		..	..	..	..	125	....	6.40	..	....	.....	Mult. R. 1, L. 2	Lobectomy	
..	..	..	..	+	..	...	....	....	..	83.8	.....	Lingula	Lingulectomy	Cured
+		+	+	—	—	138	21.6	8.20	75	77.0	.....	R.L.L. 1	Lobectomy	Improved
..	..	..	..	+	..	...	18.0	6.80	71	....	.....	R.L.L. 2	Lobectomy	Cured
..	..	..	..	+	..	...	....	6.00	..	....	.....	R.M L. mult.	Lobectomy	Cured
+	..	..	..	+	+	...	24.1	7.59	82	74.0	172/Kg.	R.M.L. mult.	Lobectomy	Cured
..	..	..	..	..	..	...	....	7.00	..	....	.....	L. lung 1	0	
..	..	..	..	..	—	...	24.9	8.79	69.7	89.2	.....	R.M.L. mult.	Lobectomy	Cured
..	..	..	..	+	..	...	....	....	..	....	.....	R.M.L.	Lobectomy	Cured
..	..	..	..	+	..	155	....	6.50	..	....	.....	R.U.L.	0	
..	..	..	..	..	..	...	....	....	..	....	.....	R.L.L. mult.	Ligation	Unchanged
..	+	..	..	..	..	...	17.0	6.50	58	....	3,800/sq. M.	R.L.L. 1	Lobectomy	Cured
..	..	..	..	+	..	...	....	....	..	....	.....	L.U.L.	.....	
..	..	..	..	..	..	96	....	6.30	..	....	.....	R.L.L. 1	Lobectomy	Death 6 mo. after operation
..	..	..	..	+	..	...	....	....	..	....	.....	R.L.L. 1	0	
..	..	..	..	+	..	Increased		....	..	....	.....	Bilat.	Lobectomy	Death in 20 hr.
..	..	..	..	+	..	...	22.0	6.90	70	74.0	4,800	R.L.L.	Lobectomy	Cured
..	..	..	..	..	..	115	18.0	5.81	..	....	.....	R.M.L. 1	Lobectomy	
..	..	..	..	..	..	...	15.5	4.80	44	....	.....	L. lung	0	
+	..	.	..	+	..	125	22.8	8.90	60	63.0	12,900; 161/Kg.	R.L.L. 4, R.M.L. 1, R.U.L. 2	Pneumonectomy	
..	..	..	..	..	..	...	....	....	..	....	Increased	L.L.L. 2	Lobectomy	Cured
..	..	..	..	..	..	107	....	5.10	..	....	.....	R.L.L. 1	Lobectomy	Cured
..	..	..	..	..	..	117	....	6.40	..	....	.....	R.L.L. 1	0	Death of brain abscess
..	+	..	..	+	..	...	19.0	8.00	..	....	.....	Mult. R.L., L.L., L.U.	3 lobectomies	Cured
—	—	—	—	+	—	90	....	4.50	..	94.0	.....	L.L. 1, R.L. 1	Lobectomy and segmental resection	Cured
—	—	—	+	+	—	102	16.0	5.50	..	88.5	.....	L.L.L., lingula, R.M.L.	Lobectomy; partial lingulectomy; partial resection R.M.L.	Cured
—	+	+	—	+	..	114	18.0	7.40	45	....	7,272	R.L.L.	Lobectomy	Cured
—	—	+	—	+	..	87	13.0	4.47	47	...	.....	R.M.L. mult.	0	

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and not particularly intelligent. His father had had, and a brother had small red "spots" on the lips. The cause of the former's death is not known; the latter was still alive and well.

The patient was well developed and nourished. His skin was typical of "farmer's skin," with pigmentation over exposed areas. Many teeth were carious, and there was severe pyorrhea. The lower lip was the site of many small (1 mm. or less)

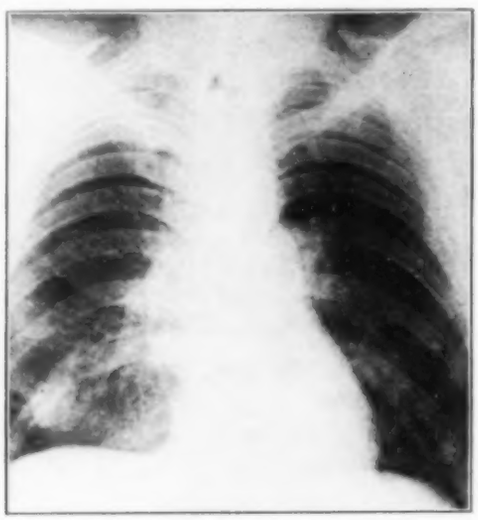

Fig. 7 (case 2).—Roentgenogram showing lobulated shadow in right lower lung field.

light red angiomas, and there were a few on the upper lip. The nasal mucous membrane was studded with similar lesions, as were the tongue, gums and buccal mucosa. There were a few pinpoint angiomas on the skin of the fingers and beneath some of the fingernails. The fingers were moderately clubbed, without cyanosis. The blood pressure was 135 systolic and 80 diastolic in the right arm and 124 systolic and 78 diastolic in the left. The heart was of normal size with mild sinus arrhythmia and a rate of 72 per minute. There was a faint systolic murmur at the apex. The most striking observation

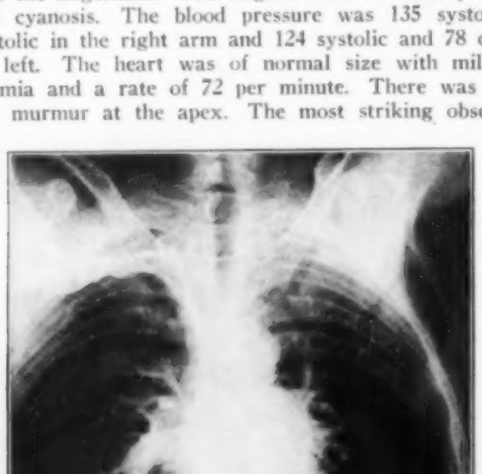

Fig. 8 (case 2).—Angiocardiogram (two and one-half seconds).

was a continuous blowing, moderately loud murmur with systolic accentuation in the fourth and fifth right intercostal spaces from the right sternal margin to the posterior axillary line, the area corresponding to that of the middle lobe of the right lung. It was loudest in the fourth interspace at the anterior axillary line, and its intensity was increased with inspiration, during which the diastolic component was more pronounced.

The hemoglobin was 13 Gm. (87 per cent). The red blood cells numbered 4,470,000 per cubic millimeter of blood, and the cell pack was 47 per cent. The white blood cell count was 9,600 with a normal differential. The sedimentation rate was 30 mm. per hour by the Wintrobe method. The Mazzini test of the blood gave a negative reaction. The bleeding time was three minutes and the coagulation time five minutes (Lee-White method). Urinalysis was normal. An electrocardiogram was interpreted as normal. A roentgenogram of the chest showed an opacity in the lower portion of the right lung field resembling a bunch of grapes, pathognomonic of a pulmonary arteriovenous varix or angioma (fig. 7). Angiocardiograms made at the Cardiovascular Research Unit of the Veterans Administration Hospital, Washington, D. C., showed this lesion more sharply and revealed smaller lesions in the right lung (figs. 8 and 9). The main lesion on the right obviously involved the middle lobe, with large vascular channels leading into and out of the angioma. The lateral view showed that the angioma was a multiple one involving a large portion of the middle lobe.

The patient was seen a few weeks later in an attack of arthralgia of the right foot. The white blood cell count was 10,400 with a normal differential. The sedimentation rate was 34 mm. per hour (Wintrobe method), and the blood uric acid 4.3 mg. per hundred cubic centimeters. The oral temperature was normal.

Fig. 9 (case 2).—Angiocardiogram (four seconds), lateral view.

The main diagnoses were palindromic rheumatism and Rendu-Osler-Weber's hereditary hemorrhagic telangiectasia with pulmonary angiomas (arteriovenous fistulas). We could think of no way in which the arthritic episodes could be linked with pulmonary varix.

COMMENT

These 2 cases are fairly representative of the others which have been reported. Operative intervention in the first case was necessary. Whether we were right in not advising surgical treatment in the second is debatable. From the standpoint of symptomatology it did not appear necessary. It was believed that if the patient began to have symptoms of hypoxemia and polycythemia surgical treatment could be instituted. The only serious cause of worry before such symptoms develop is pulmonary hemorrhage, which was fatal in 3 cases. When the fistulas are well within the pulmonary parenchyma the danger of serious hemorrhage is not as great as when they are subpleural.

Surgical treatment is the only form of treatment of value, although bloodletting gives temporary relief from polycythemia. The polycythemia, however, is compensatory and in that respect is laudable. Surgical treatment has varied from ligation of the feeder vessels and local excision to removal of two lobes of the lung and part of a third. Ligation alone is not satisfactory.

Operative intervention is well tolerated; only 2 of 26 patients died postoperatively. Table 5 lists all of the 45 cases collected and gives the important data of each.

SUMMARY

Pulmonary arteriovenous fistula is being reported with increasing frequency. Forty-three cases have been collected and 2 new ones added. The lesion is often part of the hereditary hemorrhagic telangiectasia of Rendu-Osler-Weber's syndrome. The manifestations are cyanosis, clubbing, dyspnea, polycythemia, bruit and a typical pulmonary shadow. Pulmonary hemorrhage from rupture and thrombosis from polycythemia, especially of cerebral vessels, constitute the mean complications. The treatment is surgical. If the fistulas, which are often multiple, do not occur in too many lobes this is curative.

DIAGNOSTIC VALUE OF CARDIAC AUSCULTATION

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Boston

With the rapid progress in diagnostic methods that has taken place during the past few decades in the solution of cardiac problems, there has developed an increasing degree of complexity in the technic employed by the physician. In many cases he no longer can make the proper decision on the basis of his five senses and what he carries in his simple medical kit. Elaborate instruments or tests have become necessary to solve many of the problems. Various blood tests, metabolic studies, electrocardiography, roentgenography, angiocardiography, venous catheterization of the heart and other examinations are necessary in one instance or another before the correct diagnosis can be established. These great advances that have been taking place so rapidly have not been purely of academic interest. They are absolutely essential and have enabled physicians by the application of new therapeutic procedures, medical or surgical, to treat effectively and at times to cure certain patients who formerly were beyond help. Therefore the importance of these advances cannot be minimized.

During the same period of brilliant advances, older and simpler methods of examination have suffered. This is not surprising or incomprehensible. The interest and energy expended in the development of the new have taken place at the expense and sacrifice in the application of the old. The greater the time spent in taking three electrocardiographic leads and later nine and now twelve leads, the less time is left to elicit an adequate history of the case or to auscult the heart properly. The teaching curriculum in the medical schools has become so overwhelmed with new fields of medical and correlated knowledge, that some of the old teaching and emphasis has had to be curtailed. As a result of this change, auscultation of the heart has been neglected. It is now a not uncommon experience that a patient may have a complete cardiac work-up, with roentgen examination and multiple electrocardiographic leads in which diphasic T waves and slight sagging of the S-T segment may be described, and finally may be pronounced to have "some myocardial damage," when careful auscultation in one minute would have revealed an overlooked faint diastolic murmur of aortic insufficiency or a slight presystolic murmur of mitral stenosis. The pity of it all is that the information obtained by auscultation is there for the asking, so to speak. It requires no additional expense in time or money and is available to any physician with no more tools than he always has with him.

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Read before the Section on Internal Medicine at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 8, 1949.

DECISIONS BASED ON AUSCULTATION

What is the diagnostic value of auscultation of the heart? There are three different lines of thought that the physician must go through when making decisions from auscultation. When one has acquired deserved confidence in auscultation there are certain possible diagnoses, that otherwise would have to be entertained, which can readily be dismissed. This is an aid in what might be called the destructive or eliminative aspect of differential diagnosis. At times it is extremely important to know what a patient does not have, even if it is not known what the actual diagnosis may be. The second decision that often can be made by auscultation is the determination of a definite positive diagnosis on the basis of certain observations. There are numerous auscultatory observations which are characteristic and some that are pathognomonic of certain conditions. The third is the detection of clues which are not of themselves diagnostic, but which make one think of diagnostic possibilities that otherwise might be overlooked.

I shall return to these three types of information to be obtained by means of auscultation and amplify the subject by illustrative experiences.

Dismissal of Certain Diagnoses.—If the physician has attained confidence in his auscultation and after careful examination fails to hear any murmurs whatever, even with the patient turned to the left lateral position or sitting upright, he can fairly safely eliminate the possibility of subacute bacterial endocarditis or chronic rheumatic valvular heart disease. In such circumstances there will be exceptions, as there are to all useful guides in clinical medicine, but they will be rare. I have seen instances in which subacute bacterial endocarditis was supposed to be present in the absence of any murmurs, only to find a clear aortic diastolic murmur and be able to demonstrate it to the examiner who had previously overlooked it. However, in several instances in which prolonged fever and sweats were thought to be due to bacterial endocarditis and no murmur at all could be heard, I ventured to state that some other cause for the symptom would be found. Tubercle bacilli were later discovered in the sputum samples. I must admit that on exceedingly rare occasions bacterial endocarditis was present when I could hear no murmurs. Furthermore, when following a patient who had rheumatic fever in the past, the one important observation is the presence or absence of murmurs. If such a patient is examined one or more years after the bout of rheumatic fever and murmurs are not heard, it is safe to say that the heart has escaped any important permanent damage. In fact, if there is any kind of a murmur, especially a diastolic but even a systolic, one can either be sure he is dealing with organic valvular disease or at least he cannot dismiss that possibility. It is clear, therefore, that no matter how ill or how well the patient may be or how inconspicuous the physical abnormalities may be, auscultation is the one method that often eliminates the possible diagnosis of valvular disease.

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Establishment of a Definite Diagnosis.—Pericardial Friction: The second and more important contribution that may be derived from auscultation is the establishment of a definite diagnosis on the detection of certain abnormal conditions. The palpation of a mass in the lower part of the abdomen may be indicative of a fibroid of the uterus, an ovarian cyst, a pregnant uterus, a full bladder or some tumor. Localized rales in the lungs may be due to tuberculosis, neoplasm, pneumonia, bronchitis, congestion, pulmonary infarction or a variety of other conditions. But the detection of pericardial friction almost invariably means acute pericarditis. I recall an instance in which the discovery of a faint pericardial friction sound was the sole observation that saved a physician from performing what would have proved to be an unnecessary and hazardous abdominal operation. All the features of the case pointed to an acute condition of the upper part of the abdomen that should have required an immediate exploratory operation. The most probable diagnosis appeared to be a perforated peptic ulcer, but the slight pericardial scratch was the telltale sign pointing to the heart as the cause of the severe epigastric pain.

Apical Murmur: In the overwhelming majority of cases the presence of an apical rumbling murmur,

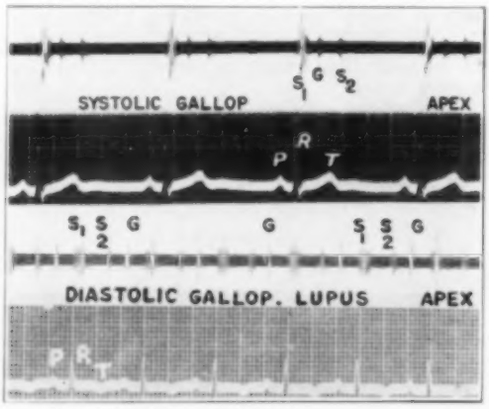

Fig. 1.—Upper tracing shows normal systolic gallop. Note that the extra sound (G) comes between a normal first sound (S_1) and a normal second sound (S_2). The subject was a normal healthy man aged 34. Lower tracing shows abnormal diastolic gallop. Note that the extra sound (G) comes between S_2 and S_1. The patient had lupus erythematosis with myocardial involvement.

diastolic or presystolic in time, is diagnostic of mitral stenosis. When one becomes familiar with the character of this murmur its detection establishes the correct diagnosis even if it is faint or present only after effort or with the patient lying on his left side. The same is also true of a faint early diastolic murmur at the base of the heart in the diagnosis of early aortic insufficiency. In some cases there will be no reliable method apart from auscultation which will aid in the diagnosis of either slight mitral stenosis or aortic insufficiency, for the roentgenograms and electrocardiograms may reveal essentially normal conditions and even symptoms of heart disease may be absent or equivocal.

Systolic Murmur: It is not to be inferred that the detection of cardiac murmurs always leads to a decisive diagnosis. There are many instances in which the murmurs are so characteristic that no other evidence is needed to be certain of the diagnosis. There are many other instances, especially when systolic murmurs are involved, in which proper evaluation may be extremely difficult. This is not the occasion to discuss the intricate problem of the interpretation of systolic murmurs. It is well known that systolic murmurs may be heard in both valvular and nonvalvular disease of the heart and may be present in patients who are suffering from noncardiac disease or no disease at all. This does not detract from the value of auscultation, but rather presents the physician an additional challenge to correctly analyze the significance of physical observations. In general it may be said that faint systolic murmurs (grade 1 or 2) may be heard with or without heart disease but louder ones (grade 3 or more) will seldom be present in normal persons and usually will indicate some form of heart disease.

Discovery of Diagnostic Clues.—The third general type of information to be derived from auscultation of the heart is what one might call clues to diagnosis. A patient may be stricken with severe epigastric pain resembling acute pancreatitis or perforated peptic ulcer, and the heart may show a classic diastolic gallop. This would lead one to suspect that the heart is the cause of the pain and would be followed by electrocardiographic studies which might confirm the diagnosis. Not infrequently the detection of a snapping first sound while the heart rate was very rapid and grossly irregular in the absence of any murmurs led me to suspect the diagnosis of mitral stenosis. Only later when the rate had slowed after digitalis therapy did the characteristic diastolic murmur become audible. At times a hyperactive first sound, not explicable otherwise, may arouse one's suspicion that thyrotoxicosis is present. I recall instances in which there was little else to make me think that the patient had hyperthyroidism. There are other auscultatory phenomena such as the effect of deep breathing, carotid sinus stimulation or an exercise test which enables the physician to make certain presumptive deductions that can later be more accurately confirmed by graphic methods. An example of this sort is the observation of an exact doubling of the heart rate from 70 to 140 after brief effort. This would make one strongly suspect the presence of auricular flutter with an auricular rate of 280.

Arrhythmias: Apart from the evidence of valvular and pericardial disease that may be obtained by simple auscultation, the detection of arrhythmias or changes in the quality of the heart sounds is important. It is evident that nowadays the widespread use of the electrocardiograph, and especially the availability of direct writing apparatus which makes the information immediately available, has made it less imperative to diagnose the various cardiac irregularities by the use of the stethoscope. The advantages, however, of quick bedside methods of auscultation are obvious, for thereby much time and expense can be saved. With increasing experience it has become possible to recognize or to suspect the presence of most of the disturbances in cardiac rhythm by simple bedside auscultation. Unfortunately, much of this valuable information is overlooked and not utilized.

Gallop Rhythm: In considering the quality and intensity of the heart sounds and the significance of changes in the character of the sounds, there is a woeful lack of understanding among many physicians. The presence of a tripling of sounds (that is, a gallop rhythm) is still too frequently overlooked. When the extra or third sound of a gallop can be identified as occurring in diastole, it almost always indicates fairly grave heart disease. What is not sufficiently appreciated is that there are gallops in which the extra sound occurs during systole that are comparatively benign (fig. 1). In fact, a midsystolic gallop is not infrequently detected in persons who do not have heart disease.

Generally it is possible to distinguish the one type from the other by auscultation. Only occasionally will it be necessary to employ graphic methods to make this differentiation.

INTERPRETATION OF FIRST HEART SOUND

What is more important is the confusion that prevails concerning the significance of the loudness and character of the first heart sound. I have asked many physicians, even specialists in the field of cardiology, what inference they draw from the detection of a muffled or exceedingly weak first heart sound. Almost invariably the reply is that it denotes a poor heart muscle, with the connotation that grave heart disease is present. Mere recollection of one's daily experience ought to throw doubt on this conception. Many patients are observed to have various forms of advanced cardiac disease, even with gross congestive failure, who display perfectly good or even loud first heart sounds. Contrariwise, others who have faint and occasionally inaudible first sounds seem quite fit. This problem deserves more detailed discussion.

Both heart sounds may be decreased in intensity if there is intervening tissue or space between the heart and the outside of the chest wall. Patients with severe emphysema, large pericardial effusion or extreme obesity may have faint heart sounds (fig. 2). That is because even normal heart sounds will be fainter to the ear after traversing a greater distance. Likewise in moribund states, when the heart is beating feebly, the sounds may be decidedly decreased in intensity. In the foregoing circumstance both heart sounds are similarly muffled and there is no selective effect on one of the sounds.

The situation is entirely different when the first heart sound is decreased and the second is not similarly affected (fig. 2). The foregoing factors cannot be the cause, as they could not produce a discriminating effect on only one of the sounds. What produces the first sound, and what determines its intensity? It is due mainly, if not entirely, to the closure of the mitral and tricuspid valves. If there is a muscular component produced in the myocardium it is practically inaudible. I have on many occasions listened to heart beats when certain cycles showed inaudible or barely detectable first sounds (with clear second sounds) that produced little, if any, vibration even in the phonocardiographic tracings, and yet the pulse for that beat was normal (second tracing, fig. 2). In other words, the strength of the contraction producing that extremely feeble first sound was just the same as that of other contractions producing first sounds five or ten times as loud which occurred several seconds later. (Compare second with last beat in middle tracing of fig. 3.) There must be some other influence at work besides the vigor of ventricular systole that profoundly affects the loudness of the first sound.

The abruptness or quickness of ventricular systole affects the first sound. When the contraction is hyperactive, as may occur in hyperthyroidism (fig. 2) or anemia, during the effort or emotion, or with certain fevers (especally acute rheumatic fever) the first sound is often accentuated. The valve leaflets close more abruptly, and a brisk sound results. Fluoroscopic examination in these circumstances not infrequently may show a "snapping" type of beat. In most of these conditions it is now well known that the velocity of blood flow is accelerated. The situation may be compared to the dull sound produced when a loose handkerchief is made taut slowly and the sharp sound when the same maneuver is performed abruptly.

The observations of Wolferth and Margolies [1] and particularly of Dock [2] have thrown further light on the changes in the intensity of the first sound. At first it was observed that in complete heart block, when the ventricles are beating very slowly and regularly and the auricles more rapidly, regularly and independently, the first sound varies greatly in intensity [3] (fig. 3). Then it was noted that there was some relationship between the auriculoventricular interval (P-R interval) and the loudness of the first sound.[1] The first sound was loudest when the P-R interval was exceedingly short, about 0.08 second, and was generally faint at intervals greater than normal, 0.22 to 0.3 second. From these and other observations Dock suggested not only that the position of the auriculoventricular valves at the moment of ventricular systole determined the intensity of the first sound but that the sound was entirely valvular in origin and had no muscular component. From these studies it has been inferred that when the mitral and tricuspid leaflets are wide apart or deep and low in the ventricular cavity the instant the ventricles contract, the sound produced as they close will be different (probably louder) than when they are nearer together or at a higher level. If the ventricles contract soon after the auricles the sound will therefore be loud; if there is delay between auricular and ventricular systole the valves will have had a longer opportunity to float upward and nearly close, and the first sound will be faint.

CLINICAL VALUE OF INTERPRETATION OF FIRST HEART SOUND

Entirely apart from the foregoing theoretic considerations, as a matter of tested clinical experience, attention to the intensity of the first sound as heard at the apex of the heart will often enable the observer to estimate the length of the P-R interval and to recognize conditions in which there is a short P-R interval, others with a prolonged P-R interval (first degree heart block) and those in which this interval is varying, such as second and third degree heart block (figs. 2 and 3). In fact, detection of these changes in the intensity of the first sound is the only reliable bedside method of making these diagnoses. If the heart rate is regular and the first sound decidedly decreased while the second sound is normal, one can be fairly certain that the P-R interval will be at least at the upper limit of normal or delayed systole (fig. 2, second tracing). Similarly, if the rate is regular and slow (30 to 40) and the first sound changes in intensity with sudden loud explosive sounds (bruit de canon) one can be certain that complete heart block with dissociation of the auricles and ventricles is present (fig. 3). This diagnosis can even be made when the ventricular rate is within the normal range (50 to 70) if the foregoing auscultatory sign is present. The usefulness of this simple auscultatory sign is well illustrated by the fact that a young house officer made an immediate diagnosis of complete heart block in a patient with acute coronary thrombosis, who showed a regular heart rate of about 60 because the first sound illustrated this peculiarity called bruit de canon. The diagnosis was immediately confirmed by electrocardiograms. One certainly would not ordinarily think about complete heart block with

1. Wolferth, C. C., and Margolies, A.: The Influence of Auricular Contraction on the First Heart Sound and the Radial Pulse, Arch. Int. Med. **46**: 1048 (Dec.) 1930.

2. Dock, W.: Mode of Production of the First Heart Sound, Arch. Int. Med. **51**: 737 (May) 1933.

3. Strazhesko, N. D.: Adams-Stokes Disease, Russ. Vrach. St. Petersburg **5**: 603, 1906.

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a heart rate within normal limits, yet simple auscultation revealed the true type of disturbance in the cardiac mechanism.

Another illustration of the value of noting the character of the first sound is the following experience. A man about 50 years of age came to the hospital because he had had a fainting spell. He felt well otherwise and was able to do his ordinary work. Physical examination revealed entirely normal conditions. The blood pressure was normal. The heart was not enlarged, and there were no murmurs or irregularities. Several physicians had examined the heart without detecting anything remarkable. When I suggested that they listen to the heart again and pay particular attention to the first sound, they only then observed that it was almost inaudible. When asked what that finding might signify, they all replied that it might mean that the heart muscle was weak. The

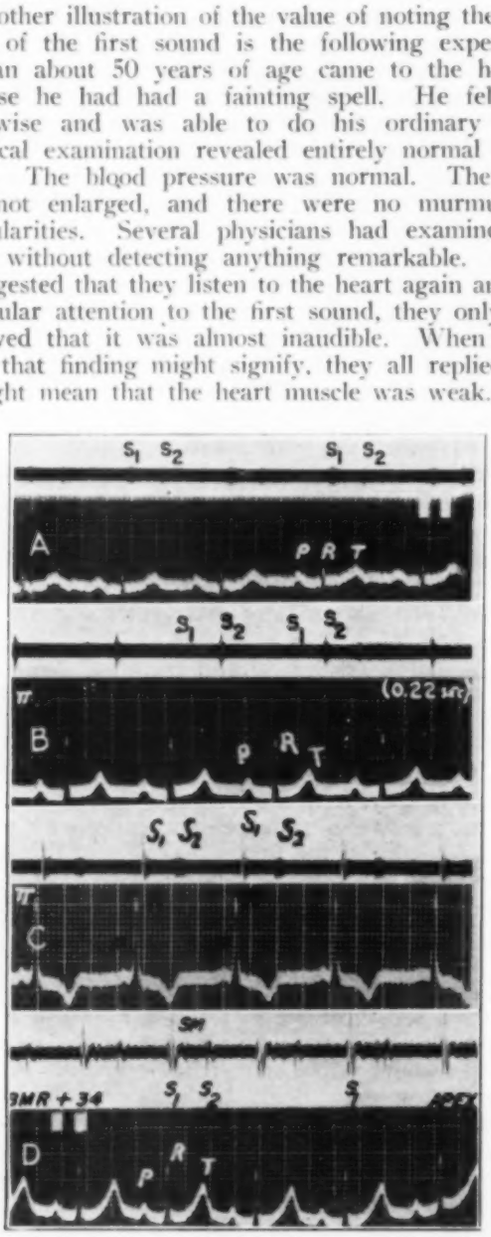

Fig. 2.—Tracing *A* shows pronounced decrease in intensity of both sounds (S_1 and S_2), in a case of severe pulmonary emphysema in a man aged 55. Tracing *B* shows almost complete absence of first sound (S_1) with good second sound (S_2) in a woman aged 24 with no heart disease; the P-R interval is slightly prolonged to 0.22 seconds. Tracing *C* shows accentuated S_1 and exceedingly short P-R interval (0.08 second) in a woman aged 24 with hypertension but no cardiac difficulty. Tracing *B* shows accentuated S_1 in a girl aged 20 with thyrotoxicosis. Note slight systolic murmur (SM).

patient, however, was strong and had no breathlessness or pain in the chest on effort. Applying the reasoning previously discussed herein, I suggested that he might well have first degree heart block with a delayed P-R interval. The electrocardiogram which was immediately taken showed the P-R interval to be 0.22 second. This not only confirmed the clinical diagnosis but threw light on the patient's complaint. Anyone with conduction difficulty might well have had an Adams-Stokes attack (syncope due to arteriosclerotic heart disease). It was later learned that he had been previously studied and found to have temporary complete heart block. There are several important inferences to be drawn from such an experience. First, abnormalities in the first sound (like other auscultatory phenomena) will not be detected unless deliberately looked for. Second, it is an error to interpret a weak first sound, when the second sound is normal, as indicative of a so-called "weak heart muscle." Finally, it is possible to recog-

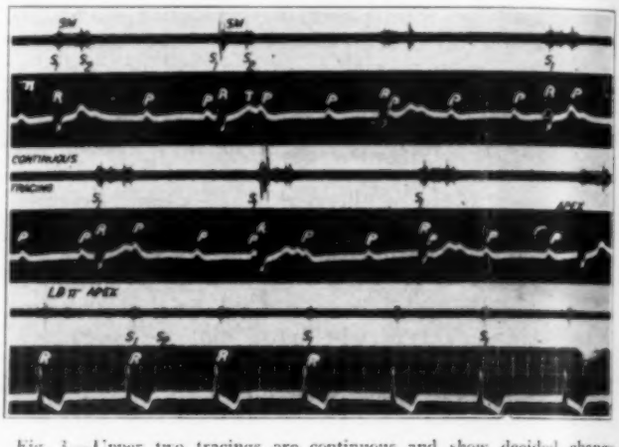

Fig. 3.—Upper two tracings are continuous and show decided change in intensity of the first heart sound (S_1) in a man aged 33 with complete heart block. Note that S_1 is loud when the P-R interval is short and faint when P-R is long. In the last cycle in the second strip S_1 is absent (only the systolic murmur is seen). Lowest tracing shows constant first heart sound (S_1) in a man aged 72 with complete block and auricular fibrillation or idioventricular rhythm.

nize, or at least to suspect, first degree heart block by simple auscultation.

If a changing time relationship between auricular and ventricular systole alters the intensity of the first heart sound, then any condition in which the auricles and ventricles are not contracting in constant relationship with each other should produce changing intensity of the first sound. This occurs in complete heart block. It should follow that when there is complete block and the auricles are fibrillating the first sound ought to be constant and not vary, for then auricular systole does not occur and would not affect the position of the auriculoventricular valves. That this is true is well illustrated in figure 3. Furthermore, there ought to be a difference between the heart sounds in ordinary paroxysmal auricular tachycardia and in

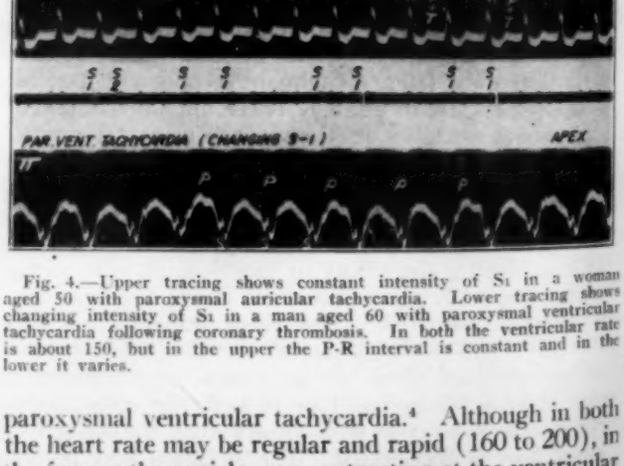

Fig. 4.—Upper tracing shows constant intensity of S_1 in a woman aged 50 with paroxysmal auricular tachycardia. Lower tracing shows changing intensity of S_1 in a man aged 60 with paroxysmal ventricular tachycardia following coronary thrombosis. In both the ventricular rate is about 150, but in the upper the P-R interval is constant and in the lower it varies.

paroxysmal ventricular tachycardia.[4] Although in both the heart rate may be regular and rapid (160 to 200), in the former the auricles are contracting at the ventricular

4. Levine, S. A.: The Clinical Recognition of Paroxysmal Ventricular Tachycardia, Am. Heart J. **3**: 177, 1927.

rate and at a constant short interval before the ventricles. That should result in first heart sounds of uniform intensity. In the latter, the auricles generally contract independently and at a slower rate than the ventricles, which should result in a changing intensity of the first sound. Actual clinical experience bears out these theoretic predictions (fig. 4).

The distinction between the ventricular and the auricular type of paroxysmal tachycardia which one may make by simple auscultation is exceedingly important. It may not be possible to obtain an electrocardiogram, and the condition may require immediate attention. Furthermore, most of the procedures that are effective in controlling auricular tachycardia are valueless in the ventricular type. Finally, ventricular tachycardia is generally a desperate complication of a serious underlying cardiac condition and requires prompt treatment. Digitalis, which is so commonly employed in cardiac emergencies with exceedingly rapid heart rates, will be not only useless but harmful in this condition, whereas quinidine is often effective and may be life saving. There will be times when the first suspicion that the patient has paroxysmal ventricular tachycardia will be derived from simple auscultation.

I have cited only a few examples of auscultatory observations that generally can be readily made by any physician with a simple stethoscope. There are many others which need not be discussed here. The purpose of this communication is to revive interest in this ancient method of examination, because much useful information at present is being overlooked by the members of the medical profession.

SUMMARY

There are many auscultatory observations which at present are overlooked or misinterpreted that can easily be made and properly analyzed by any physician by the simple use of the stethoscope. Some of these observations are practically pathognomonic, and others are strongly suggestive of certain cardiac diagnoses that otherwise would not be made. The decision can often be made in a few minutes, and occasionally the condition is critical and requires immediate institution of therapy. It is urged that medical teachers and practitioners pay more attention to the ancient art and science of auscultation of the heart.

ABSTRACT OF DISCUSSION

DR. HOWARD B. SPRAGUE, Boston: It is wise of Dr. Levine to bring back to attention the value of the accuracy of diagnosis by simple methods. The methods which Dr. Levine has noted are of extreme importance. I would like to stress what he says about the possibility of ruling out what the patient does not have. This would eliminate perhaps 50 per cent of the possibilities with respect to diagnosis. Obviously, one must remember that the most serious types of heart disease, including coronary disease with angina pectoris, cannot be diagnosed by any method except by listening to the patient's story. A pericardial rub may be the only definite clue to obscure illness, either the transient rub of rheumatic infection or the more persistent rub of the so-called idiopathic pericarditis. The ability to recognize the normal third heart sound is of great importance because it prevents overdiagnosis of heart disease, and training in this recognition enhances one's ability to discover the middiastolic murmur of left ventricular dilatation which comes directly after the third heart sound in many cases of rheumatic carditis and which may disappear with recovery. It also improves one's critical analysis of the pathologic gallops, the commonest of which is an exaggeration of the third sound mechanism. The range of intensity of the normal first heart sound is great, measured in terms of energy, as was shown by Williams and Dodge years ago. The amount of energy recorded in the loudest normal first heart sound which they examined was forty times that of the weakest normal first heart sound. These sounds may be of low frequency, and the ear may not interpret them as loud sounds even though they have considerable intensity. Therefore, I wish to second Dr. Levine's statement that weakness of the first heart sound, or rather of both sounds, is not commonly a manifestation of cardiac weakness. The statements that he made about the variation of the loudness of the first heart sound indicated an important clinical observation. The mechanism of this is not well explained at present, and experimental work would seem to indicate that the auricle in contraction actually tends to bring the mitral cusps together, if not in apposition, at the end of the contraction. I would stress the necessity of using both an open bell and the diaphragm stethoscope in examining all cardiac patients in order to get the discriminating effect of these two types of chest pieces. The skill in the use of the stethoscope which has been attained by Dr. Levine makes it possible to dispense with the more elaborate kit of medical tools, especially in emergencies.

PROPHYLACTIC (BCG) VACCINATION TO CONTROL TUBERCULOSIS

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Through the offices of the World Health Organization the governments of various countries have been solicited to sanction prophylactic vaccination against tuberculosis, through the use of BCG vaccine, of 15,000,000 children and young adults who are nonreactors to tuberculin. In the United States and Canada pressure is being exerted in various sections to increase the use of this vaccine in an effort to control tuberculosis. These programs can readily gain enthusiastic lay support, simply because of their magnitude, from those without a clear understanding of the problem of tuberculosis. Any dissension about the concept behind this approach to the control of tuberculosis must be objective. The present discussion of the use of BCG vaccine will be on a factual basis.

SAFETY

One of the chief claims of those now promoting the use of BCG vaccine is that it is harmless. All investigators who have studied the problem firsthand can agree that this living culture of bovine tubercle bacilli will not produce progressive tuberculosis provided that it is properly prepared, thoroughly controlled and properly given. The safeguards now in effect in the preparation and use of BCG vaccine are to be highly commended; however, it is essential to keep in mind that this relatively nonpathogenic strain had its origin from a highly pathogenic bovine type of tubercle bacillus and that, until such time as the factors governing the pathogenicity of tubercle bacilli are known, BCG vaccine must not be considered as a virus fixé.

The sponsors of mass prophylactic vaccination must rely heavily on the hope that the increased resistance to tuberculous infection will be sufficient to protect against future infection, for under present conditions exposure to infection is unavoidable. It is this phase of the vaccination program that creates concern relative to the eventual effectiveness of any prophylactic vaccination against tuberculosis.

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Read before the Section on Diseases of the Chest at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 8, 1949.

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EFFECTIVENESS

Thorough studies on experimental animals have already been conducted to determine the effectiveness of BCG vaccine, and the facts obtained must not be disregarded. It has been demonstrated conclusively that increased resistance to infection with virulent tubercle bacilli is produced through vaccination; however, this resistance is not complete. The most favorable results that I obtained were that 5 per cent of the vaccinated animals subsequently infected with virulent bacilli died from progressive tuberculosis as quickly as though prophylactic vaccination had not been given; 45 per cent showed small, slowly progressing tuberculous lesions, and lesions did not develop in 50 per cent. I conducted one experiment with cattle which illustrates the difficulty of assessing the prophylactic effect of BCG vaccination. Twelve of 24 steers of approximately the same age were vaccinated with BCG, and 12 were kept as controls. Nine months after vaccination 2 vaccinated animals were killed, and the only evidence of tuberculosis was a necrotic focus at the site of vaccination. Every animal, control and vaccinated, was then given 10 mg. of virulent bovine tubercle bacilli intravenously. Within six weeks 3 animals, 2 controls and 1 vaccinated, succumbed to fulminating generalized miliary tuberculosis. During the ensuing nine months no more animals died, and all were then killed. Postmortem study showed that every animal had small tuberculous lesions in the lungs and other organs and that it was not possible to determine from either the gross or microscopic nature of the lesions which animals had been vaccinated. The difficulties of assessing the value of vaccination with BCG in experimental animals under controlled conditions emphasize the greater difficulties encountered in experiments in man because of the existence of uncontrolled and uncontrollable conditions.

PATHOGENESIS AND EPIDEMIOLOGY OF TUBERCULOSIS

It has long been taught that tuberculosis is a family disease and that young children and young adults especially are affected. This is unfortunate, for it is generally believed that if tuberculosis fails to develop before persons reach 40 years of age, they will not contract the disease. This belief has led to an emphasis of the problem of tuberculosis in young persons and to an absence of concern over the much more serious problem of tuberculosis in adults beyond the fourth decade of life, as demonstrated by the present program for mass vaccination with BCG of millions of children and young adults who do not react to tuberculin. Tuberculosis cannot be combated effectively without a thorough consideration of the pathogenesis and epidemiology of the disease.

Infection with tuberculosis is greatest where masses of human beings are congregated within limited areas. It is a herd disease that depends for its propagation on the passage of tubercle bacilli, most often through the air, from one person to another. Recent experimental studies have demonstrated that droplets of air containing tubercle bacilli must be less than 15 microns ($\frac{1}{2,000}$ of an inch) in diameter before they can penetrate the alveolar spaces of the lung and start an infection. Indeed, the tuberculous process has a small beginning.

An examination of the problem of tuberculosis in metropolitan New York, the largest and most congested city in the country, is most revealing. The data contained in the vital statistics of the United States indicate that during the past half-century a great change for the better has occurred in the mortality rate in tuberculosis. For present consideration data from the vital statistics of the city of New York for the years 1905 and 1945 will be used, although it is recognized that all such data contain errors of diagnosis that can hardly be canceled simply because a large volume of data can be utilized. Between the years 1905 and 1945 the number of deaths from all causes increased 9 per cent and the number of registered deaths from tuberculosis decreased 64 per cent. Although the population showed a decided increase during these four decades, the total number of deaths decreased 82 per cent among persons less than 15 years of age and 60 per cent in the age group 15 to 40 years of age but increased 128 per cent in persons over 40 years of age. Deaths from tuberculosis in these same age groups decreased 91, 77 and 28 per cent, respectively. These phenomenal changes occurred without any program of mass vaccination against tuberculosis, and it is difficult to determine the actual effect of any single measure that has been used against the disease.

It is understandable that the seriousness of tuberculosis in young children should be emphasized, for once it has gained a foothold, especially in babies less than 1 year of age, it is a rapidly progressive and highly fatal disease. However, even forty years ago such deaths accounted for only a small percentage of the total deaths for tuberculosis. In 1905 in the city of New York 37 per cent of all deaths occurred among persons less than 15 years of age, and of these only 4 per cent died from tuberculosis. Only 11 per cent of all deaths from tuberculosis occurred in children under 15 years of age, largely in young infants, whereas 31 per cent occurred among persons over 40 years of age. In 1945 deaths from tuberculosis in persons less than 15 years of age accounted for only 3 per cent of all such deaths, while 61 per cent occurred among persons over 40 years of age. There is no intention to detract from the tragedy of tuberculosis in infants, yet it is evident that the problem of tuberculosis in adults is far more serious. Furthermore, it is understood generally that the prevalence of tuberculosis in children is related directly to the disease in adults.

TUBERCULOSIS IN ADULTS

A statistical statement used frequently and effectively is that today tuberculosis ranks first among diseases as a cause of death in the age group 15 to 35 years. The significance of this statement cannot be evaluated properly except in relation to other facts. In this age group during the last four decades there occurred in New York City a decrease of 60 per cent in the number of deaths from all causes and 77 per cent in the number of deaths from tuberculosis. In 1905 58 per cent of all registered tuberculous deaths occurred in persons between the ages of 15 and 40 years, whereas in 1945 the percentage was only 36. In spite of this phenomenal change tuberculosis still ranks first among all diseases as the cause of death in this age group. This merely indicates that tuberculosis has not decreased as much as have other diseases. Vital statistics of the United States for the year 1947 indicate that only 26 per cent of all deaths due to tuberculosis occurred in persons in the age group 15 to 35, whereas 70 per cent occurred in older adults. It is evident that the problem of tuberculosis in young adults is linked to the problem in older adults. An oversimplification and a

distorted view of the problem of tuberculosis results whenever emphasis is given to the rank of this disease as a cause of death in any specified age group.

Formerly it was held that progressive tuberculosis among adults resulted from recrudescence of a primary tuberculous infection contracted in early childhood. Tuberculin surveys, clinical investigations and careful studies of data compiled at necropsy reveal the fallacy of this dogma. Progressive tuberculosis in young adults represents largely a primary infection acquired in adult life. Hence, prophylactic vaccination with BCG now is urged for young adults. Since increased resistance to tuberculous infection may be gained through vaccination, it may seem both unwarranted and unwise to inject a note of caution at this point. However, of those who acquire a primary infection, or what might be called a natural vaccination, in adult life only a small percentage ever manifest clinical signs of the disease. Furthermore, there is no guaranty that either an artificial or a natural vaccination will prevent the development of a serious reinfection later in life.

REINFECTION

In the December 1948 issue of the *American Review of Tuberculosis* I have pointed out that in adults over 40 years of age, especially in men, both minimal pulmonary tuberculosis and deaths from tuberculosis are caused in large part by reinfection after a previous infection had healed.[1] In spite of a natural vaccination with virulent tubercle bacilli, a new progressive disease may develop. It is this situation that to date has received little attention in programs aimed at the control of tuberculosis. Certain facts contained in vital statistics deserve serious thought. Beyond the age of 40 years deaths from tuberculosis are three times greater among men than among women, and for the country as a whole almost three fourths of all deaths from tuberculosis occur in this age group. Tuberculosis cannot be effectively controlled until the problem of tuberculous reinfection has been met, and it is because of this situation that prophylactic vaccination against tuberculosis as now advocated would seem to be a rather puny weapon.

Tuberculosis is most difficult to evaluate because it seldom occurs in epidemic form and because it has a slow tempo of development in adults. This is shown clearly in the experience of the Metropolitan Life Insurance Company. In this organization all applicants for employment are given a preemployment roentgenologic examination of the chest, and those who evidence pulmonary tuberculosis are not employed. The average age at the time of employment is 20 years, and after employment a yearly roentgenologic examination of the chest is made. Starting with as tuberculosis-free a group as it was possible to assemble, 226 new cases of tuberculosis were discovered between the years 1935 and 1946. Only 7 per cent of these new cases were found in the first year after employment, whereas 65 per cent were discovered more than five years after employment. Of these, almost half had been employed more than ten years. In 90 per cent of cases, careful questioning of these persons with new tuberculous infections failed to reveal any contact with a person known to have tuberculosis. These data were obtained in a nonvaccinated group of adults. It is evident that an enormous task confronts those who anticipate conclusive proof of the value of prophylactic vaccination with BCG on a long-range basis.

It is difficult to obtain decisive results in any program of prophylactic vaccination against tuberculosis. It is extremely doubtful that artificial vaccination can produce results superior to natural vaccination with small numbers of living virulent tubercle bacilli, and yet natural vaccination fails to control the disease. It appears illogical to base a major attack against any disease on a skirmish against a minor phase of the problem. (This is asking the tail to wag the dog.) It is suggested that greater emphasis be given to the major problem in tuberculosis—unrecognized tuberculosis and that due to reinfection in adults over 40 years of age. An effective solution to this problem would make the use of prophylactic measures in youth unnecessary.

SUMMARY

The chief concern in mass prophylactic vaccination is not the vaccine but rather the implications relative to the control of tuberculosis. Since the population is mobile, it is illogical to restrict vaccination to the slums or to certain selected groups of young adults such as nurses and physicians, for other young persons are also exposed to infection and should receive the benefit of this procedure. If every person less than 25 years of age in the United States had been vaccinated with BCG in 1946 and if perfect protection had been attained (a doubtful premise), in what manner could the vital statistics on tuberculosis have been affected in 1947? If there had been no deaths from tuberculosis in persons less than 25 years of age in 1947 only one seventh of the registered deaths from this disease would have been erased, and it seems probable that the majority of deaths remaining in the records would have been related to disease acquired subsequent to the complete healing of a natural vaccination. One need not disagree with the concept of vaccination as a procedure to hold that an effective control of tuberculosis cannot be obtained without a major attack on the source from which the disease is spread. The present program of mass vaccination with BCG does not touch this problem, and it would appear illogical to anticipate that artificial vaccination could succeed where natural vaccination has failed.

ABSTRACT OF DISCUSSION

DR. ROBERT J. ANDERSON, Washington, D. C.: The effect of tuberculosis on the people of this country and the world warrants consideration of any potential factor that might aid in the control of this disease. Although the tuberculosis death rate in this country had been depressed to 33 per 100,000 population in 1947, almost 50,000 persons died from tuberculosis. Dr. Medlar has depicted the changing effect of tuberculosis among persons of various ages. Proportionately, more and more of the deaths from tuberculosis are occurring in middle-aged and elderly persons and fewer and fewer in young persons, with declining death rates for all ages. This observation indicates the necessity for careful study of the groups to whom BCG vaccine or any other immunizing agent might be given, for with an adequate control program it should be possible to be rationally selective in determining the groups to receive vaccination. This will also require careful study of the tuberculin sensitivity levels of the population geographically, socially and by age groups. The effect of the vaccine on man must be studied in terms of changed levels of tuberculin sensitivity, of duration of sensitivity and effectiveness and of the occurrence of pulmonary tuberculosis as physicians know it. Conflicting observations and conclusions in the several fields touching on the problems of prophylactic vaccination necessitate more investigative work. The effect

1. Medlar, E. M.: The Pathogenesis of Minimal Pulmonary Tuberculosis. A Study of 1,025 Necropsies in Cases of Sudden and Unexpected Death, Am. Rev. Tuberc. **58**: 583-611 (Dec.) 1948.

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of BCG vaccine on man and its contribution to health must be measured carefully in the same circumstances which produce tuberculosis in some persons and leave others untouched. This study should produce opinions well grounded in fact. The extensive foreign programs now well under way may result in such information. In this country, however, the facts should be sought deliberately. Dr. Medlar pointed out the difficulties of doing such work with tuberculosis. But these are difficulties which must be overcome. I urge an intensification of control efforts in seeking out persons with tuberculosis, rendering them noncontagious through isolation and treatment and reestablishing them as normal members of society. BCG vaccination does not possess the property of producing absolute immunity and can only be contributory to the control of tuberculosis. Danger lies in the creation of a false sense of security for those who are vaccinated with BCG unless the technics which lessen the hazards of contracting tuberculosis are practiced rigorously.

DR. HENRY C. SWEANY, Chicago: It has been a quarter of a century since Calmette reported his rather inconclusive results with BCG vaccination. Since then there has been little enthusiasm for its use outside of France, Scandinavia and Latin America. The reason lies in the many uncertainties in such a vaccine and in the difficulties of establishing proof of its stability and effectiveness. Recently, new interest has been aroused by the work of Holm in Denmark and Aronson, Ferguson, Rosenthal and others in this country. The best argument in favor of vaccination with BCG seems to be that natural resistance gained from small infections has protected the race from devastating epidemics of the disease. There is little doubt that a completely healed infection protects against another infection. However, this protection is only relative and varies greatly not only in the time that it persists but probably also in its quantitative aspects. The chance of contracting the disease a second time no doubt varies with the severity of the infection from which the patient has recovered. It is rare to find tuberculosis developing in a person who has made a complete recovery from one attack of the disease. BCG vaccination, however, cannot be expected to afford the same protection as an attack of the disease. Still it will give some protection. It is probably valuable when used to give relative protection to uninfected persons, such as nurses and technicians, who work with or come in contact with tubercle bacilli. In other situations it may be useful in inverse proportion to the effectiveness of public health measures, as in primitive, thickly populated and war-torn countries. I would also add, in homes where it is impossible to isolate infants from persons who are carriers of the tubercle bacillus. There are situations, however, in which BCG vaccine should not be used. Except in persons who have been exposed to the disease, it should not be used in places with a low morbidity rate such as exists in Iowa and Minnesota because the methods in use are solving the problem, and vaccination with BCG invalidates the best methods of case finding and diagnosis by creating a tuberculin-positive population. Finally, there are possibilities of overemphasizing a BCG vaccination program to the detriment of the regularly established methods of control. To use any system that tends to supplant, hamper or delay the present methods used in finding the reservoirs of infection and administering treatment to those with tuberculosis is not good public health practice.

War Burns.—In the last war, burns came next in frequency and severity to orthopedic injuries. In the next war they will probably outnumber all other injuries. In the Royal Air Force in this war probably more than 22,000 men were incinerated with or without other serious injuries. However, 4,500 burned air crew men were recovered from crashed planes or parachuted in flames to safety. Of that number, 3,600 had sustained burns of the hands and of the face. This is a relatively new disease, a disease of wartime which we have called the Airman's Burn. . . . The treatment of this condition which appeared suddenly in 1941 with the Battle of Britain entailed the abandonment of coagulative therapy advised at the outset of hostilities and the institution of an open form of treatment.—Sir Archibald McIndoe, London, England, *Postgraduate Medicine*, September 1949.

SPONTANEOUS NONTRAUMATIC SUBDURAL HEMATOMAS

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Virchow,[1] in 1857, was one of the first to recognize the difference between traumatic and spontaneous subdural hemorrhage.[2] He thought that the latter arose from repeated small hemorrhages of a chronically inflamed dura, and to this condition he gave the name "pachymeningitis haemorrhagica interna." Many since have attempted to evaluate the role of infection in this lesion. Putnam,[3a] who, in 1925, was the first to review this problem thoroughly, observed that infection had not been proved except as a rare etiologic factor. Leary [4] also expressed the belief that the inflammatory cellular reaction is "incidental or accidental."

Baker,[5] in 1938, supported Virchow's views, but he offered no additional proof for the infection theory, and it appears that Putnam's conclusions still hold.

Chronic alcoholism was suggested as a cause of subdural hemorrhage by Kremiansky [6] in 1868. Leary [4] performed autopsy in 50 cases of subdural hemorrhage. In 27 of these there had been addiction to alcohol. He expressed the belief that a person subject to alcoholism is more prone to head trauma, but later stated: "Alcohol and 'C' avitaminosis may be a factor in many cases of massive subdural hemorrhage following trauma which is in most instances minor." Putnam and Putnam [3b] concluded from a survey of the literature that a subdural hematoma had not been produced experimentally by chronic alcoholic intoxication.

Subdural bleeding has been attributed to ascorbic acid ("vitamin C") deficiency. Ingalls [7] reported 5 cases in infants, 3 with scorbutic changes in the bones. He expressed the belief that an underlying bleeding diathesis could be postulated on the basis of scurvy but that the free subdural bleeding was produced by a definite or an insignificant head trauma causing rupture of a bridging dural vein and that organization and removal of the clot were impeded by the scorbutic process, with the result that a neomembrane surrounded free blood.

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Read before the Section on Nervous and Mental Diseases at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 8, 1949.

1. Virchow, R.: Haematoma Durae Matris, Verhandl. d. phys. med. Gesellsch. zu Würzburg **7:** 134-142, 1857.

2. There is a difference of opinion concerning the anatomic location of subdural hemorrhage. J. A. Hannah (The Etiology of Subdural Hematoma: Anatomical and Pathological Study, J. Nerv. & Ment. Dis. **84:** 169-186, 1936), Baker,[5] Kaump and Love,[22] and H. C. Trumble (Chronic Subdural Hematoma, M. J. Australia **34:** 106, 1947) expressed the belief that traumatic or spontaneous chronic subdural hemorrhage is intradural (into the layers of the dura). However, according to T. Leary (Subdural or Intradural Hemorrhages? Arch. Path. **28:** 808-820 [Dec.] 1939), Trotter [37] and most investigators, massive acute and chronic hemorrhage is subdural and the common source of bleeding a ruptured vein traversing the subdural space. Leary stated that if the arachnoid is broken, the hemorrhage is usually absorbed and that an intact arachnoid appears to be a necessary element in the production of chronic subdural hematoma.

3. (*a*) Putnam, T. H.: Chronic Subdural Hematoma: Its Pathology, Its Relation to Pachymeningitis Hemorrhagica and Its Surgical Treatment, Arch. Surg. **11:** 329-393 (Sept.) 1925. (*b*) Putnam, T. J., and Putnam, I. K.: The Experimental Study of Pachymeningitis Haemorrhagica, J. Nerv. & Ment. Dis. **65:** 260, 1927.

4. Leary, T.: Subdural Hemorrhage, J. A. M. A. **103:** 897-903 (Sept. 29) 1934.

5. Baker, A. B.: Subdural Hematoma, Arch. Path. **26:** 535-559 (Aug.) 1938.

6. Kremiansky, J.: Ueber die Pachymeningitis interna haemorrhagica bei Menschen und Hunden, Arch. f. Path. Anat. **42:** 129-161; 321-351, 1868.

7. Ingalls, P. A.: Role of Scurvy in the Etiology of Chronic Subdural Hematoma, New England J. Med. **215:** 1279-1281 (Dec.) 1936.

Ingraham and Matson [8a] found clinical evidence of "vitamin C deficiency" in 20 of their 98 cases of subdural hematoma in infants. This deficiency could not be considered a uniform or a common finding. They and others [8b, c] believed that head trauma is the important etiologic factor.

Suter [9] expressed the opinion that thiamine deficiency (B_1-hypovitaminosis) may cause subdural bleeding, but there is no confirmation of this theory.

Spontaneous subdural hemorrhage caused by a blood dyscrasia such as hemorrhagic purpura needs no documentation.

Huegenin [10] stated that in 10 per cent of his cases spontaneous subdural hemorrhage was accompanied by pernicious anemia. This high percentage has not been reported by other observers.

Subdural bleeding due to thrombosis of cerebral veins or of the cerebral dural sinuses has been recorded by Bailey and Hass,[11] Tischer,[12] Bucey and Lesemann [13] and me.[14] Miscellaneous causes such as carcinomatosis and sarcomatosis of the dura,[15] violent coughing, syphilis, tuberculosis, rupture of a small cerebral aneurysm,[16] vascular malformation and hemorrhage over a brain tumor have been reported.[16c] A list of conditions causing nontraumatic subdural hemorrhage is given in the accompanying outline.

A discussion of the etiologic factors of spontaneous subdural hemorrhage would be incomplete without an evaluation of the role of *trivial* head trauma in its production.

Trotter,[17] in 1914, disagreed with Virchow and favored the traumatic origin of this lesion. Trotter's view is generally accepted today.[18]

However, Baker [5] expressed the belief that the etiologic background of subdural hematoma is quite variable and that, although trauma may be important in many cases, one must be guarded in accepting a trivial injury as an important cause. King [16b] supported the theory that even in traumatic cases there may be a predisposing factor such as short veins and friability of the vessel walls. Although Munro [19] held that trauma is most important, he agreed that the chronic solid hematomas can be caused by scurvy and blood dyscrasia. Groff and Grant [20] expressed the opinion that there remains in the case of the chronic form of hematoma some underlying factor or factors which necessarily precede the precipitating trauma and that there are numerous instances in which trauma plays no part and the hematoma is said to develop spontaneously. These authors reviewed 15 articles on subdural hematomas, covering a majority of the reports up to 1942. They eliminated single case reports and tabulated a total of 223 cases. It is noteworthy that, although most of the articles were captioned "traumatic subdural hematomas," only 165, or 74 per cent, of the cases were definitely of traumatic origin whereas in 58, or 26 per cent, no history of injury was recorded.

Kunkel and the late Walter Dandy [21] reported a group of 48 cases of subdural hematoma and stressed the fact that in 17, or 36 per cent, no history of even trivial trauma could be elicited despite their "constant interest in this phase of the lesion."

Kaump and Love,[22] after studying 30 postmortem specimens of subdural hemorrhage, 13 of traumatic and 17 of spontaneous type, concluded, "No evidence could be deduced from this series of cases which would put all cases of so-called subdural hematoma into one etiological classification; that is, traumatic or spontaneous." Kaump and Love remarked that since the history is often unreliable and since the etiologic role played by mild trauma is well recognized, this would "tend to make one feel that they may well have all been of traumatic nature."

Leary [4] also was of the opinion that subdural hematomas are practically always the result of injury to the head but that the injury may appear trivial and rarely is severe enough to cause fracture. However, a review of Leary's statistics shows that 54 per cent of his cases concerned persons who were addicted to alcoholic liquors and possibly suffering from ascorbic acid deficiency and that in at least 16 per cent of his entire series there was no history of head trauma.

Although most observers have stated that the blow to the head may be trivial and that the symptoms may come on days, weeks, months or even years after the trauma, there are many who would regard these long latent intervals with suspicion so far as the relation of the head trauma to the cause of the symptoms is concerned.[23] Laudig, Browder and Watson [16a] reported 143 cases of subdural hematoma. In 101 of these cases the injury to the head resulted in immediate unconsciousness. Munro remarked, "We may conclude that the patients suffering from subdural hematoma will present a history of injury to the head followed in practically every case by some degree of unconsciousness and very often headaches." He then went on to say, "However, the latent interval may vary up to years."

Henschen [24] published a series of 121 cases of subdural hematoma, in 30 of which the latent interval

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12. Tischer, H.: Differential Diagnosis of Eclampsia: Case of Sinus Thrombosis and Pachymeningitis Haemorrhagica, Zentralbl. f. Gynäk. **66:** 1717-1722 (Oct.) 1942.

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16. (*a*) Laudig, G. H.; Browder, E. J., and Watson, R. A.: Subdural Hematoma: One Hundred and Forty-Three Cases Studied in Five Year Period, Ann. Surg. **113:** 170-191 (Feb.) 1941. (*b*) King, J. E. J.: Extradural and Subdural Hematomas, Am. J. Surg. **59:** 248-271 (Feb.) 1943. (*c*) Voris, H. C.: Subdural Hematoma, J. A. M. A. **132:** 686-692 (Nov. 23) 1946.

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19. Munro, D.: Cerebral Subdural Hematomas: A Study of Three Hundred and Ten Verified Cases, New England J. Med. **227:** 87-95 (July 16) 1942.

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was less than a day, in 77 less than a week and in 103 less than three weeks. Voris,[16c] who reported 100 cases of subdural hematoma, remarked, "Any injury which occurs more than three months before the onset of symptoms is regarded with suspicion." Putnam stated:

> . . . There is something very mysterious about the occurrence of these lesions and their delayed relation to trauma. . . . If such clots were likely to follow trauma, one would expect to find them more often after fractures of the skull, and they would be a frequent postoperative complication of intracranial osteoplastic procedures (in which subdural clots are fairly rare).

He concluded:

> . . . It would appear, therefore, that in the disorder under consideration there must be some individual predisposition of the meninges favorable to the subdural seepage of blood.

From the citations it appears that one is justified in questioning the significance of minor blows to the head as a cause of spontaneous subdural hemorrhage unless a blood dyscrasia or an abnormality of the cerebral vasculature is proved.[25]

Publications limited to spontaneous subdural hemorrhage are rare. Occasional cases can be found by reviewing articles on traumatic or chronic subdural hematoma. Munro [26] mentioned an acute type in a 59 year old woman with a blood pressure of 220 systolic and 110 diastolic. The patient went swimming, complained of severe headache and fainted. When hospitalized ten days later she had paralysis of the left side of the body. For at least a year before the onset of the acute illness her systolic pressure was only 135. The patient died after a large clot had been removed from the right subdural space.

Laudig and associates,[16a] King [16b] and Voris [16c] have also described cases of acute subdural hematoma due to ruptured aneurysm. Acute subdural hemorrhages caused by thrombosis of cerebral veins or dural sinuses have been reported by Bailey and Hass,[11] Tischer [12] and Bucey and Lesemann.[13]

The latest report of cases of spontaneous subdural hemorrhage is that by Feld [27] in 1947 in the French literature. He collected 15 cases. The patients were 11 males and 4 females; 6 were between 15 and 30 years of age and 9 between 45 and 60 years. Fourteen of his patients were operated on; 9 recovered and 6 died. The blood pressure, the blood urea nitrogen and the bleeding and coagulation times were normal in all cases. He reported 2 cases of acute hemorrhage, due in one to the ingestion of an anthelmintic (male fern) and in the other to rupture of an aneurysm of the sylvian artery. He observed that spontaneous subdural hematomas are usually unilateral and occur over the surface of a hemisphere and never at the base or in the interhemispheric space. He described the membrane histologically as inflammatory tissue composed of zones rich in fibroblasts, newly formed capillaries and zones of lymphoplasmocytic infiltration. He found varices of the dura mater in 2 cases and expressed the belief that these anomalies situated along the course of the perforating dural veins are the most likely source of spontaneous subdural hemorrhage. The cause of the launching of the attack is incidental and may be intercurrent disease or trauma.

25. Putnam [3] and L. Jores (Ueber die Beziehungen primären subduraler Blutungen zur Pachymeningitis haemorrhagica interna, Verhandl. d. deutsch. path. Gesellsch. **1**: 49, 1898) expressed the belief that a traumatic hematoma may be distinguished from a spontaneous one by microscopic examination of the membrane. K. Inglis (Subdural Hemorrhage, Cysts, and False Membrane: Illustrating Influence of Intrinsic Factors in Disease When Development of Body Is Normal, Brain **69**: 157-194 [Sept.] 1946), R. G. Cobientz (Chronic Subdural Hematomas, Surgery, **4**: 194-210 [Aug.] 1938), Baker [6] and Kaump and Love [22] held that the slight difference observed microscopically did not warrant this etiologic distinction.

26. Munro, D.: The Diagnosis and Treatment of Subdural Hematoma, New England J. Med. **210**: 1145-1160, 1934.

27. Feld, M.: Contribution à l'étude des hématomes sous-duraux spontanés, Rev. neurol. **79**: 97-108, 1947.

ETIOLOGIC CONSIDERATIONS IN SPONTANEOUS NONTRAUMATIC SUBDURAL HEMORRHAGE

1. Infection (Virchow 1857)
   - A. Acute
     1. Systemic
     2. Meningitis
     3. Exanthems (coughing)
     4. Thrombophlebitis of cerebral veins and dural venous sinuses
   - B. Chronic
     1. Syphilis
     2. Tuberculosis
2. Chronic alcoholism (vitamin deficiency?)
3. Avitaminosis
   - A. Ascorbic acid ("vitamin C")
   - B. Thiamine ("vitamin B," according to Suter [9])
4. Hemorrhagic diathesis
   - A. Blood dyscrasia
     1. Pernicious anemia
     2. Hemophilia
     3. Scurvy
     4. Purpura
5. Malformations of blood vessels
   - A. Aneurysms of cerebral arteries
   - B. Varices of cerebral and dural veins
6. Miscellaneous
   - A. Carcinomatosis and sarcomatosis of dura
   - B. Hemorrhage from brain tumor
   - C. Exogenous toxins
     1. Anthelmentic (male fern) (Feld [27])

REPORT OF CASES

Five cases of spontaneous subdural hemorrhage are reported from a series of 100 consecutive cases of subdural hematoma in which I have operated. Cases of subdural hygroma, as well as any case in which there was a suggestion of even trivial head trauma, were excluded from the series.

The cases could be divided into two clinical groups: Group 1: Acute subdural hemorrhage with the symptoms of a cerebrovascular accident. Group 2: Chronic subdural hemorrhage with the symptoms of a slowly expanding intracranial lesion.

Group 1: Spontaneous subdural hemorrhage with the symptoms of a cerebrovascular accident.

Case 1.—M., a white man aged 66, a clergyman, had intermittent headaches, vertigo and vomiting for several weeks, became irrational and was admitted to the medical service of Dr. J. C. Doane of the Jewish Hospital on June 21, 1948. His rectal temperature was 101 F.; pulse rate, 20; respiratory rate 32, and blood pressure, 160 systolic and 80 diastolic.

He had not previously had vascular hypertension, and his history disclosed no hemorrhage, head trauma or alcoholism.

He was stuporous and had nuchal rigidity. The left pupil was dilated, the right was small, and both did not react to light; the eyegrounds were normal. The patient showed decerebrate rigidity.

The blood-tinged spinal fluid was under a pressure of 260 mm. of water. The red blood cell count and the hemoglobin content were normal. There were 17,500 white cells, of which 92 per cent were polymorphonuclears and 8 per cent lymphocytes. The thrombocyte count was 332,000. The urine contained albumin (1 plus) and 2 to 4 granular and hyaline casts per high power field. The bleeding and clotting times were 2 minutes. The blood urea nitrogen was 11 mg. per hundred cubic centimeters. The icterus index was 15 and the van den Bergh test for serum bilirubin showed 6.3 units. The total blood protein was 6.2 Gm. per hundred cubic centimeters. The Wassermann test of the blood was negative. A diagnosis was made of bilateral intracerebral hemorrhage, more on the left side, and the lifting of an osteoplastic flap on the left revealed a huge subdural clot. A fine stream of blood was seen pumping from a pinpoint opening in the left sylvian artery, and this was considered the source of the subdural hemorrhage. Examination of the artery did not reveal any aneurysm or obvious pathologic change. The bleeding was stopped with "gelfoam" pledgets. There was no evidence of any intracerebral bleeding. A burr opening over the right posterior frontal area showed no subdural hemorrhage.

The patient improved slightly, but forty-eight hours later he became moribund, with a rectal temperature of 107 F. The osteoplastic flap was reopened and a small extradural hemorrhage removed. The cerebrum was edematous. Cannulation showed no intracerebral bleeding, and a decompression was done. A burr opening was made over the right prefrontal area, and exploration showed no subdural or intracerebral hemorrhage.

The pathologist's report of the blood clot follows: "A large amount of blood clot, approximately 6 cm. in diameter, was examined. In one portion there appeared to be a membrane. Microscopic section showed blood clot with a little fibrin and a few polymorphonuclear leukocytes" (Dr. H. Ingelby).

The patient's stormy course with eventual convalescence included jacksonian convulsions, pulmonary atelectasis, pulmonary abscesses, persistent rectal bleeding of unknown cause and epididymitis. The scope of this paper does not permit a detailed description of the vigorous manner, tenacity and judgment with which Dr. Doane and his staff applied various life-saving therapeutic measures in combating these complications.

On discharge, November 2, five months after admission, the patient was out of bed. There was partial anomia with occasional jargon-like speech. Right homonymous hemianopsia was present, and there was excellent return of power in the right upper and lower extremities. There were slight spasticity and weakness of the left upper and lower extremities.

He returned to the hospital approximately three weeks later because of tingling and numbness in the left arm and leg. The blood pressure was 132 systolic and 90 diastolic. Speech had improved, and the decompressed area was soft. Right homonymous hemianopsia and partial left superior temporal anopsia were present. Slight paresis was still evident on the left, with hyperactive reflexes and a Babinski sign. There was no impairment of sensation. The right side had entirely recovered. The spinal fluid was blood tinged, and the pressure was 170 mm. of water. The blood coagulation time was 10 minutes and the prothrombin time 70 per cent of normal. It was thought that the patient had a slight subarachnoid hemorrhage over the right half of the cerebrum, but he recovered promptly.

In May 1949, approximately one year after the onset of his original symptoms, examination revealed progressive improvement in speech. The patient walked into the office with a slight limp. His blood pressure was 140 systolic and 90 diastolic. The neurologic examination showed no change.

Comment.—This patient had attacks of dizziness, occipital headaches and vomiting for a period of two weeks and then suddenly became irrational. On admission the spinal fluid was blood tinged and the neurologic examination showed involvement of both cerebral hemispheres, but more of the left. Operation revealed a massive subdural hemorrhage over the left cerebral hemisphere which appeared to have come from a pinpoint opening in the left sylvian artery. There was no history of vascular hypertension, and during hospitalization the blood pressure never rose above 120 systolic. There was no history of blood dyscrasia, although three sisters were said to be bleeders. The patient's bleeding and coagulation times were high normal. The resident remarked that there had been unusual oozing from the skin incision during exposure of the saphenous vein of the foot, and about eight days after the initial operation the patient had considerable hemorrhage of obscure origin from the lower bowel for a period of one week.

The white blood cell count of 17,500 cells with 92 per cent polymorphonuclears, the presence of a small amount of albumin and 2 to 3 granular and hyaline casts in the urine and the elevated rectal temperature of 101 F. suggested the possibility of some infectious process which had weakened the blood vessel wall. Periarteritis nodosa was also considered.

CASE 2.—Mrs. A. S., a white woman aged 51, was admitted to the medical service of Dr. Louis Soloff of the Temple University Hospital on Sept. 8, 1948. On September 1 the patient had fainted while washing clothes; she promptly recovered and had persistent headaches for seven days until the date of admission, when she became unconscious. The rectal temperature was 101 F.; the pulse rate, 70, and the respiratory rate, 28. The blood pressure was 148 systolic and 70 diastolic on admission but went as high as 194 systolic and 100 diastolic during her stay. The patient had been "hypertensive" for many years; there was no history of head trauma or alcoholism.

She was semistuporous and had nuchal rigidity. There were partial left hemiparesis and a bilateral Babinski sign. Petechiae were present on forehead and face.

The grossly bloody spinal fluid was under a pressure of 270 mm. of water. The red cell count and the hemoglobin content were normal. The white cell count was 19,050, with polymorphonuclears 84, lymphocytes 11 and monocytes 5 per cent. The urine contained albumin (slight trace) and sugar. The Wassermann test of the blood was negative. The bleeding time was 1 minute; the coagulation time, 3½ minutes. The sedimentation rate was 22 mm. in one hour; the blood urea nitrogen was 16 mg. per hundred cubic centimeters.

The roentgenograms of the skull revealed no abnormality. A preoperative diagnosis was made of spontaneous hemorrhage involving the right frontal lobe of the cerebrum. A right frontotemporal exploration was done thirteen days after the onset of the symptoms, and a large subdural clot was evacuated. The source of bleeding was not found. The cortical area representing the arm center appeared slightly full, but cannulation did not reveal intracerebral hemorrhage. A microscopic examination of the specimen was reported as revealing pure blood clot with no evidence of membrane (E. E. Aegerter).

There was considerable improvement in the hemiparesis, and the blood pressure was 150 systolic and 100 diastolic on discharge, September 30.

The patient was reexamined by Dr. Soloff in April 1949 (seven months after onset of symptoms). She was comfortable; her blood pressure was 160 systolic and 90 diastolic, and she had recovered the use of her left arm and leg with only slight residual spasticity. The fundi showed right optic atrophy of obscure cause.

Comment.—There was no history of blood dyscrasia. The patient was known to have high blood pressure. The only noteworthy laboratory findings were the marked elevation of the white cell count associated with fever and the increased sedimentation rate. Although these reactions could have been due to the subdural hemorrhage, it was also thought that some type of infection may have weakened a blood vessel wall, the hypertension being a precipitating factor.

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Group 2: Spontaneous subdural hemorrhage associated with symptoms suggesting a slowly expanding intracranial lesion.

Case 1.—A. G., a white man aged 32, was admitted to the neurosurgical service at the Temple University Hospital May 24, 1942, complaining of right retro-orbital pain and vomiting of two weeks' duration. The temperature, the pulse rate and the respiratory rate were normal. The blood pressure was 130 systolic and 80 diastolic. There was slight bilateral sixth nerve weakness with bilateral choked disk of 1 to 2 diopters. There was marked left hemiparesis. The red blood cell count was 5,915,000. The hemoglobin content was 15 Gm. The white cell count was 14,350, with polymorphonuclears 77, lymphocytes 14, monocytes 7 and eosinophils 2 per cent. The thrombocyte count was 354,000. The coagulation time was 3½ minutes. The Rumpel-Leede test produced a moderate number of petechiae. The sedimentation rate was 20.5 mm. in one hour. The urinalysis gave entirely normal results. The spinal fluid pressure was 260 mm. of water, and the fluid was clear. The Wassermann tests of blood and spinal fluid were negative.

Roentgenograms of the skull revealed that the pineal gland was displaced slightly dorsad and caudad to the normal site; an encephalogram showed the lateral ventricles displaced to the left, and it was suspected that there was a brain tumor in the right frontal area.

On June 4 two burr openings were made in the right frontal and the parietal area, respectively. They revealed a capsule about 3 mm. thick and an underlying chocolate-colored liquid subdural hematoma, which was evacuated.

The microscopic examination of the membrane was reported as follows: "Chronic granulation tissue with fibrosis; Infiltrating mononuclear inflammatory cells with recent and old hemorrhage, and new capillary proliferation" (E. E. Aegerter).

Recovery was uneventful. Questioning of patient and family revealed no history of head trauma or alcoholism, but the patient recalled that he had a persistent cold before the onset of his headaches. He said that he rarely drank orange or tomato juice, but there were no obvious signs of scurvy.

Examination six and a half years later showed complete recovery. His blood pressure was 132 systolic and 80 diastolic.

Comment.—A spontaneous subdural hemorrhage occurred in a young healthy man. There was no evidence of vascular hypertension, blood dyscrasia, trauma, syphilis or alcoholism. The only suggestion of infection was a "cold" at the onset of his illness and an elevation of the white cell count and the sedimentation rate on admission. However, the last two findings could have been a systemic reaction sequent to the subdural bleeding. Ascorbic acid deficiency was also considered.

Case 2.—T. T., a white man aged 60, was admitted to the neurologic service of Dr. Sherman Gilpin Jr., at the Temple University Hospital on Dec. 19, 1944. He was partially aphasic and had suffered from headaches and vomiting for five months, attributed to a heat stroke during the summer. There was no history of head trauma, alcoholism or hemorrhage. The rectal temperature was 99.8 F.; the pulse rate, 80; the respiratory rate 22. The blood pressure was 140 systolic and 90 diastolic.

The eyes were normal. There was right hemiparesis with a bilateral Babinski sign.

Cyanosis of the face, the lips and the nail beds of unknown cause had been present for three months. He had received sulfadiazine for two months before admission because of his "sinus condition." A blood sulfadiazine level taken on admission was 2 mg. per hundred cubic centimeters, and two spectrometer analyses showed traces of sulfmethemoglobin. A spinal puncture done elsewhere was recorded as having shown fluid under a pressure above 180 mm. of water and containing 78 cells, with no differential count being made.

The red cell count and the hemoglobin content were normal; there were 9,100 white cells, of which 83 per cent were polymorphonuclears, 12 lymphocytes and 5 monocytes. The urine, the blood urea nitrogen, the coagulation time, the prothrombin time and the ascorbic acid level were all normal. The thrombocyte count was 340,000, and 196,400 when repeated three days later. The Rumpell-Leede test produced no petechiae. The sedimentation rate was 20 mm. in one hour. The result of the Wassermann test of the blood was not reported. The icterus index was 6, and the van den Bergh test, direct, for serum bilirubin showed 0.25 mg. per hundred cubic centimeters.

Roentgenograms of the chest revealed no abnormality. A cardiac consultation gave no explanation for the cyanosis. Roentgenograms of the skull showed that the pineal gland had shifted 8 mm. to the right of the midline. A provisional diagnosis was made of tumor or abscess of the left temporofrontal lobe.

On December 22 a burr opening was made in the left frontal and parietal area. A subdural capsule, 3 mm. in thickness, was opened, and approximately 60 cc. of chocolate-colored liquid blood was removed. The patient improved for five days and then gradually became stuporous. A left frontoparietal osteoplastic flap was reflected twelve days after the first operation. An unusually vascular dura was exposed and was then peeled off the underlying greenish blue membrane of a massive subdural hematoma. After removal of the hematoma, an additional underlying hygroma was evacuated. The patient died eight days later. An autopsy showed a large extradural hemorrhage at the site of the surgical intervention. The source of the bleeding could not be found, but it was assumed that it came from a dural vessel. The autopsy gave no clue to the cause of the cyanosis or of the original subdural hematoma. There were no hemorrhagic lesions in any of the organs and no evidence of syphilis. The cause of death was postoperative extradural hemorrhage associated with multiple pulmonary abscesses and bronchopneumonia.

The microscopic examination of the membrane and clot removed at the first operation was reported as follows: "The clot is well organized in its peripheral and central portion. It is densely infiltrated by chronic inflammatory cells and by pigment-laden macrophages. There are endothelial and fibroblastic proliferation. Much calcification is seen throughout. Giant cells of the foreign body type are identified lining up around crystalline material. The dura is little changed from the normal except for fibrinous thickening. The subdural membrane is composed of edematous fibrous tissue showing fibrinoid regeneration, patchy calcification and scattered blood pigmentation. The endothelial lining cells are hyperplastic and form a fringelike substance. In one corner there is a massive necrotic debris. Occasional psammoma-like bodies are seen. Diagnosis: chronic organized subdural hematoma" (A. T. Peale).

Comment.—This patient had chronic headaches, which were attributed to a previous heat stroke and later to sinusitis. He also had undiagnosed cyanosis of face, lips and finger nails which antedated the use of a sulfonamide compound. Cardiac examination and autopsy offered no explanation for the cyanosis. There was no history of vascular hypertension, bleeding diathesis, alcoholism or head trauma. Although the extradural hemorrhage was attributed to the surgical intervention, the same factors which produced the subdural bleeding may have contributed to its occurrence.

Case 3.—C. W., a white man aged 59, was referred to the neurosurgical service of Dr. J. E. Gotwals April 27, 1945. The patient complained that he had suffered from vomiting and right supraorbital headaches for one month. The temperature and the pulse and respiratory rates were normal. The systolic blood pressure readings varied between 132 and 140. There was no history of head trauma or alcoholism. Four years previously the patient had urinary retention and hematuria. This improved, but recently he had noticed urinary frequency and incontinence. The neurologic examination revealed nothing except for increase of the left deep tendon reflexes.

The spinal fluid was normal except that the Wassermann reaction was weakly positive on two occasions. The Wassermann test of the blood was negative. The complete blood count was normal. The urine showed a trace of albumin and 50 to 60 red blood cells per high power field but no casts. The blood urea nitrogen was 15.6 Gm. per hundred cubic centimeters. The bleeding, coagulation and prothrombin times and the blood level of ascorbic acid were normal. The thrombocyte count was 301,700. The sedimentation rate was 18 mm. in one hour.

Roentgenograms of the chest showed healed, obsolete tuberculosis. Retrograde pyelography and intravenous urography revealed advanced involvement of the right kidney. A Mantoux test was moderately positive. Roentgenograms of the skull revealed that the pineal gland had shifted 4 mm. to the left of the midline. A provisional diagnosis was made of tumor of the right frontal lobe possibly metastatic from the kidney. Exploratory craniotomy was done on the right side, May 7, and a liquid and solid subdural hematoma was evacuated. A bleeding vein was found in the premotor area. Whether it was the original source of the subdural hemorrhage was impossible to decide, since it could have been torn during the removal of clot. The cortex in the region of this vessel appeared somewhat full, but a subcortical incision showed no evidence of tumor. The patient's convalescence was uneventful. The microscopic appearance of the hematoma was described as follows: "The tissue consists of a large blood clot surrounded by a relatively cellular fibrous tissue membrane. In some areas this membrane has a pronounced lining of cells that appear to be endothelial in origin. Beginning organization of the clot is visible. Diagnosis: organizing hematoma with no evidence of tumor tissue."

The patient was discharged on May 21. He refused any treatment of the suspected lesion of the kidney. Three years later examination showed no return of cerebral symptoms.

Comment.—This patient presented right frontal headaches of five weeks' duration associated with vomiting. There was no history of head trauma, alcoholism, blood dyscrasia or vascular hypertension. Although the Wassermann reaction of the spinal fluid was slightly positive on two occasions, the Wassermann reaction of the blood was negative, and there was no history of any previous syphilitic infection. There was a history of past hematuria, and tuberculosis or tumor of the right kidney was suspected but not demonstrated. There was no evidence of any intracerebral tuberculosis or neoplasm.

RÉSUMÉ

The etiologic factors of spontaneous subdural hemorrhage are reviewed. A classification of the causes of this type of hemorrhage is presented. The etiologic significance of trivial head trauma is discussed.

Five cases of spontaneous subdural hemorrhage are reported. The patients were 4 men and 1 woman. The youngest (a man) was 32 years of age, and the remainder were from 51 to 66 years of age. The hematoma was unilateral in all. The symptoms were acute in 2 and chronic in 3. One patient died from postoperative extradural hemorrhage, which possibly was of the same cause as the original subdural hemorrhage. The remaining 4 recovered.

Although the site of hemorrhage was found in only 1 case (a pinpoint opening in the sylvian artery) and the cause demonstrated in none, the following facts may be of clinical interest:

1. Only 1 patient had vascular hypertension. None had a history of alcoholism.
2. There was debatable evidence of syphilis in 1 case; in the remainder the Wassermann reactions were negative.
3. The coagulation time was normal in all cases, and the bleeding time was normal in 4 cases in which it was observed.
4. Low intake of ascorbic acid was admitted in 1 case.
5. The white blood cell count was elevated in 3 cases.
6. The sedimentation rate was elevated (average, 20 mm. in one hour) in the 4 cases in which it was determined. In none was there anemia. The rectal temperature was normal in 3 cases. The white blood cell count was normal in 2 and elevated in 2.

CONCLUSIONS

1. Spontaneous nontraumatic hemorrhage was found in 5 of 100 consecutive patients operated on for subdural hemorrhage.

2. Spontaneous nontraumatic subdural hemorrhage may occur in two clinical forms: (1) acute, with symptoms of cerebrovascular accident; (2) chronic, with symptoms of a slowly expanding intracranial lesion.

3. The source of the bleeding is rarely detected at operation or on inspection of the gross specimen. It may be a bridging vein on the way to a dural venous sinus, a perforating dural vein or a dural venous varix. In 1 patient in this series the bleeding was from a pinpoint opening in the sylvian artery.

4. The etiologic factors of the pathologic change involving the blood vessel are usually obscure. Infection, vitamin deficiency, alcoholism, syphilis, have all been accused but rarely proved to be etiologic.

5. The role of trivial head trauma as a cause of subdural hematoma has been given undue prominence in the literature. It is rarely a direct cause of the hemorrhage but may be a precipitating cause if previous anatomic abnormality or disease of the cerebral vasculature exists.

6. The causation of spontaneous subdural hemorrhage could be more accurately established if the following were carefully investigated: (1) the history, by means of a comprehensive routine questionnaire; (2) the results of the physical examination and laboratory tests for evidence of blood dyscrasia, avitaminosis, infection or abnormal vasculature; (3) the operative area for abnormality of vasculature, and (4) the membrane and clot for histologic and bacteriologic evidence of infection.

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ABSTRACT OF DISCUSSION

DR. RUDOLPH JAEGER, Philadelphia: Dr. Scott has undertaken the difficult task of attempting to prove, from the literature and his own experience, the nontraumatic origin of subdural hematoma. The difficulty is evident from the fact that, after a detailed critical analysis of 100 consecutive cases of subdural hematoma, he found only 5 that fitted the clinical criteria of spontaneous subdural hematoma, and in only 1 of the 5 did he find any pathologic factor that might have a definite bearing on the cause of the hemorrhage. In one instance the patient had hypertensive disease and a discernible point of arterial rupture. No one can deny that spontaneous hemorrhage occurs from friable arteries, as can be readily observed in hemorrhages of the retinal vessels. I have also uncovered a spurting arterial rupture after wiping away a

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large clot from a sylvian artery in a case of vascular hypertension. This paper emphasizes the fact that subdural hematoma may be caused by many factors other than extracranial trauma. However, it must be admitted that spontaneous subdural hematoma is comparatively rare and that no clear clinical picture has been presented to differentiate the nontraumatic from the traumatic type. Absence of a history of trauma certainly does not preclude the possibility of a hematoma of traumatic origin. If subdural hematoma may be inflammatory in origin, why is it that no one has ever described definite organization between the dura and the capsule of the hematoma or adherence of the capsule and the arachnoid? While the dura is always adherent to the outer aspect of the capsule, I have never found that it could not be readily stripped from the underlying hematoma. I described the first 2 cases of subdural hematoma I reported (1930) as cases of "pachymeningitis hemorrhagica interna," as did Hassin prior to that, because the more appropriate terminology of subdural hematoma had not then come into vogue. In one of my cases the hematoma might well have been considered of inflammatory origin, since it was a necropsy specimen without history. In the second one, however, its traumatic origin was proved beyond a shadow of a doubt, as there had been a violent blow on the head causing a fracture of the skull. The occurrence of the multilaminate subdural hematoma is most difficult to explain on a traumatic basis. The mechanics of its formation may be explained by the enlargement of the hematoma from the absorption of cerebrospinal fluid, tearing small vessels in the dural-arachnoid trabeculations, thereby setting up other hemorrhagic layers under the initial hematoma. Thus, additional hemorrhages occur in the subdural space precisely as they do in extradural hemorrhage, but at a much slower rate.

Dr. H. C. Voris, Chicago: Three years ago, before this section in San Francisco, I reported a series of 100 consecutive cases of subdural hemorrhage, and in that group I had only 3 cases in which there was no history of significant trauma. Dr. Scott's group, in his series of 100 cases, is only slightly larger. There is no doubt that contributing factors are present even with trauma, such as vascular anomalies and tumors. My series contained examples of that. I think that Dr. Scott is a little dogmatic in his dismissal of apparently trivial trauma to the head as a possible cause of subdural hematoma. Those of you who have seen the now famous motion picture Drs. Sheldon, Pudenz and Restarski prepared a few years ago could not fail to be impressed by the movements of the brain under the lucite calvarium when the monkey was subjected to subconcussive trauma. Such movements of the brain can, I am sure, rupture short bridging veins, or anomalous bridging veins between brain and dura, and thus produce subdural hemorrhage, even though the trauma itself may seem to be insignificant.

Although there is no way of proving it, I am convinced that in some of these cases in which one does not have a history of significant trauma the patient did have an injury and either has forgotten it or is unable to recall it. All have had the experience of operating on a patient in the absence of a history suggestive of subdural hemorrhage and then, later, having the family or the patient himself recollect significant trauma. However, Dr. Scott's presentation is of great value because it teaches again that one must not fail to consider the possibility of subdural hemorrhage even though there is not a history of significant trauma.

Dr. Michael Scott, Philadelphia: Time does not permit, for example, including such cases as that of a patient who, while in the ocean, was struck on the head by a wave and five months later had headaches and then subdural hematoma. That patient was excluded from this series because of a history of trauma. The correlating of trauma to symptoms is frequently difficult in some of these cases. On the other hand, my associates and I believe that minimal trauma may be a factor if there is an associated abnormality of the cerebral vasculature.

Clinical Notes, Suggestions and New Instruments

TRAUMATIC AMENORRHEA WITH DYSMENORRHEA

JACOB MEZER, M.D.
Boston

Severe dysmenorrhea without menstrual flow is an unusual sequel to uterine curettage. It begins on the first anticipated menstrual day following curettage and may recur at monthly intervals until treatment is successful. When this condition results from obstruction of the cervical canal due to traumatic stricture with hematometra, the problem is a mechanical one requiring reestablishment of patency of the canal. This report concerns 2 patients with a similar syndrome (amenorrhea with dysmenorrhea) in which there was no obstruction in the cervical canal and in which the problem was of a different nature.

REPORT OF CASES

Case 1.—A woman aged 37, the mother of three children, complained of two attacks of severe abdominal pain without menstrual flow recurring at monthly intervals following a therapeutic abortion by dilatation and curettage at seven weeks' gestation. Two years before, dilatation and curettage had been done for menstrual irregularity. There was no previous history of dysmenorrhea. During her third attack, examination showed diffuse abdominal tenderness and an extremely tender but not contracted uterus. The patient was confined to bed for a week because of pain, which opiates relieved only slightly. Two days later the uterus was no longer tender, and a uterine sound was passed easily without encountering any obstruction or tapping a hematometra. Hysterosalpingography disclosed only slight irregularity of outline of the uterine cavity. Seventeen days later a fourth attack occurred, concurrent with the end of a biphasic basal body temperature curve. The cause of the amenorrhea was not established. Because of the remote, though unlikely, possibility of failure of endometrial regeneration, 3.75 mg. of sodium estrone sulfate were given daily for four days starting at the onset of her pain. The next ovulation was delayed by four days. Six days after ovulation endometrial tissue was removed for biopsy, which showed a secretory phase of twenty days' development. At this time, because her molimina were distressing, the estrogen therapy was repeated. Eight days after removal of tissue for biopsy, a normal flow appeared without discomfort. Thereafter, full painless flow recurred regularly.

Case 2.—A housewife aged 35 had a curettage and intrauterine packing for an incomplete miscarriage at ten weeks' gestation. Dilation of the cervix had not been necessary, because the cervix was patulous, tissue having been passed. Eight years before, dilatation and curettage were performed for metrorrhagia; and five years before, an incomplete miscarriage at three and one-half months' gestation was completed by dilatation, curettage and intrauterine packing. There had never been any dysmenorrhea. One month after her recent miscarriage her molimen was associated with severe colicky low abdominal pain of eighteen hours' duration. There was no menstrual flow. One month later this episode recurred. During the second half of her third cycle, she was given 2.5 mg. of sodium estrone sulfate daily for six days. The third attack of pain recurred concurrent with the end of a typical biphasic basal body temperature curve. In the hope of preventing a fourth attack of pain by suppressing ovulation, she was given 3.75 mg. of sodium estrone sulfate for twenty-five days, starting the first day of her fourth cycle. On the seventh day of this cycle an unsuccessful attempt at uterine sounding was made elsewhere. The basal body temperature curve this month was atypical, consistent with inhibition of ovulation. Seven days after the last dose of estrogen, a pink-tinged discharge was noted for two days. There was no pain. Three days later the cervix was too sensitive to allow manipulation. Eleven days after ovulation the cervix was sounded with the patient under

Assistant in gynecology and gynecologic pathology, Beth Israel Hospital.

anesthesia. There was no obstruction or hematometra. An endometrial biopsy showed a secretory response of twenty-five days' development. Three days later the patient began a normal, full, painless menstrual flow, with resumption thereafter of her usual cycle.

COMMENT

Stamer [1] reviewed 37 cases from the literature since 1892 and added 24 of his own. He classified them as follows: (1) complete atresia without ovulation, pain or bleeding; (2) incomplete atresia with ovulation, pain and internal bleeding resulting in hematometra, and (3) incomplete atresia with ovulation and pain but without bleeding either internally or externally. When hematometra was present, the passage of a probe or uterine sound relieved stenosis at the internal os and allowed drainage of old blood, with recovery. In incomplete atresia without bleeding, simple sounding or dilation of the cervical canal was equally effective in relieving amenorrhea and periodic dysmenorrhea, whether it had prevailed for a few months or years. Biopsies of endometrial tissue removed at the onset of menstrual flow following probing after ovulation showed a secretory response. Endocrine therapy was used in cases of anovulatory flow. Stamer expressed his belief that the syndrome is due to varying degrees of atrasia following trauma to the endometrium, especially at the internal os.

Asherman,[2] who studied 29 patients with the same syndrome as that observed in my patients, found stenosis varying from slight narrowing to complete occlusion but could not correlate the degree of stenosis with the severity of symptoms. The syndrome developed after such procedures as manual removal of the placenta, intrauterine packing, curettage performed for abortion, postpartum bleeding or hydatidiform mole; also, after repeated curettage for other reasons. In all but 2 patients, relief of stenosis by the simple passage of a sound or dilators facilitated the return of normal menstruation four weeks after the last menstrual pains, concurrent with the end of the biphasic basal body temperature curve. When the biopsy specimen was taken immediately after breaking through the atresia, cyclic phenomena could not be observed, the endometrium remaining in a "state of inactivity." Since there was no hematometra or inhibition of the hormonal cycle, he concluded that the syndrome was due not to narrowing of the internal os but to failure of the endometrium to respond to endocrine sitmulation and that the inactive endometrium could be caused to respond by local mechanical probing.

Asherman's postulate of failure of endometrial regeneration, however, is in contradiction with the observations of Stamer and the evidence in the cases reported herein that a secretory endometrium is present in these patients. Moreover, a state of inactivity is not likely on the basis of other evidence. After normal menstruation the endometrium is restored with great rapidity. Rutherford and Mezer [3] found that endometrial regeneration promptly ensued after full term pregnancy whether or not ovulation was inhibited by lactation. After spontaneous abortion, regeneration may be complete within seven days, even if followed by curettage and even if ovulation is delayed by retention of placental and decidual tissue.

Although the dysmenorrhea in this syndrome may be related to ovulation [4] in my patients, the fact that it is intimately associated with amenorrhea suggests that some other mechanism is responsible. It is possible that the trauma of curettage sometimes disturbs the process of endometrial disintegration at the end of the hormonal cycle and that sounding or removal of tissue for biopsy, or both, reestablish the normal mechanism involved. The association of dysmenorrhea with amenorrhea would suggest that abolition of either symptom must be followed by abolition of the other. Perhaps more information on this mysterious condition might be obtained by removal of endometrial tissue for biopsy at the onset of the attack of severe pain. Histologic study of such tissue may be of value in elucidating the mechanism of essential dysmenorrhea.

SUMMARY

1. Traumatic amenorrhea and dysmenorrhea is an unusual and trying sequel to uterine curettage.

2. Various theories as to causation are discussed.

3. Relief of the severe pain may be afforded by the inhibition of ovulation with the use of estrogens.

4. Recovery may follow removal of endometrial tissue for biopsy. This suggests that endometrial tissue be taken at the onset of severe pain.

171 Bay State Road (15).

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3. Rutherford, R. N., and Mezer, J.: Regeneration of Uterine Mucosa after Spontaneous Abortion and Normal Delivery, J. A. M. A. **119:** 124-128 (May 9) 1942.
4. Sturgis, S. H., and Albright, F.: The Mechanism of Estrin Therapy in the Relief of Dysmenorrhea, Endocrinology **26:** 68, 1940.

Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

AUSTIN SMITH, M. D., *Secretary.*

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SATURDAY, OCTOBER 29, 1949

SURGICAL TREATMENT OF FOCAL EPILEPSY

Horsley, in 1909, described a method of excision of an area of motor cortex for the relief of athetoid and convulsive movements of an upper extremity. Sachs, in 1935, reported observations on 11 patients on whom "The Horsley Operation" was performed. There were 9 patients cured, 1 death and 1 recurrence in these 11 cases of Jacksonian epilepsy. Furlow,[1] in 1938, reported on 16 more patients from the same clinic with 2 deaths, a cure in 7 instances, improvement in 6 and no change in 1. Penfield and Erickson[2] published in 1941 an analysis of all patients who had cortical excision carried out during a ten year period, 1929-1939. One hundred and fifteen of these patients were followed from one to eleven years; 43 per cent were free from seizures or had one or two attacks; 26 per cent considered themselves 50 per cent improved; 9 per cent were slightly helped, and 22 per cent were not improved.

Cerebral seizures, as differentiated from essential epilepsy, may be caused by a variety of focal lesions, such as meningocerebral scar, local cortical atrophy, local microgyria, expanding lesions, such as brain tumor or cerebral abscess, cysts and other abnormalities. All of these factors, Penfield emphasizes, have a common feature, which is the presence of a zone or area of ganglion cells in which the circulation has been interfered with to such an extent that the life of the cells is threatened. It is at some point in these peripheral and partially destroyed zones that the actual focus in which the epileptic discharge originates is to be found. Partial anoxemia, if not sufficient to destroy, produces in ganglion cells an abnormal state in which spontaneous neuronal activity is increased. This increase may be called the epileptogenic factor. Penfield stresses that, for surgical therapy to be effective, it is not sufficient to cut adhesions, to open a cyst or, in some cases, to remove a tumor. The epileptogenic focus in the marginal partly involved gyri must also be removed. Voris[3] reported a series of 24 patients with focal epilepsy on whom cortical excision was done. Of 20 patients followed for six to eight years, 2 have had no seizures since the operation, in 6 there rarely has been a seizure, 7 are improved and 5 are not significantly better. Cortical resection produced improvement in 8 of 11 patients, as compared with similar results in 15 of 20 patients in the entire series.

In an analysis of 76 patients operated on during the six year period, 1939 to 1944, Penfield and Steelman[4] report 1 fatality which was caused by an infected extradural hematoma and meningitis. Of the 75 patients followed, cortical excision was performed in 59 and craniotomy without excision in 16. Of the 59 patients who had a cortical excision, 15 were completely free of seizures, 18 were almost completely free, 8 were 50 per cent improved, 7 were only slightly improved and 11 felt that they had derived no benefit from the operation. In 41 per cent of the patients the physical and neurologic examination pointed to a local lesion.

Roentgenography is valuable in the diagnosis of a local lesion by revealing asymmetry of the skull or cranial hemiatrophy which is strongly suggestive of birth injury. Pneumoencephalography may yield some evidence of local cerebral atrophy. Electroencephalography was practiced in all of Penfield's cases and was found to be valuable in identifying the location of epileptogenic discharge in a high proportion of cases of focal epilepsy. The activity of the focus may be increased by hydration, hyperventilation or use of metrazol® (pentylenetetrazol). Useful as it is, one cannot rely entirely on the electrographic report alone. Penfield stresses that in the 16 craniotomies without excision performed by him there was not a single successful case. The cause of the original lesion in his series was most often head injury, second, birth injuries and, third, local infection. The cause was unknown in 12 per cent of the excisions and in 62 per cent of the negative explorations. The best guide to localization of an epileptogenic zone is the seizure pattern. The greatest supplementary help a surgeon can receive is an electrographic study by an expert.

In a recent publication Penfield[5] emphasizes that a focus within the brain which gives rise to occasional epileptic seizure may also exert an adverse influence on the general brain development and brain function. This was demonstrated by a case report of a child who had a brain injury at the time of birth. He seemed to

1. Furlow, L. T.: Subpial Resection of the Cortex for Focal Epilepsy: Further Observations, J. A. M. A. **111**: 2092-2095 (Dec. 3) 1938.
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3. Voris, H. C.: Surgical Treatment of Epilepsy, Dis Nerv. System **4**: 363 (Dec.) 1943.
4. Penfield, W., and Steelman, H.: The Treatment of Focal Epilepsy by Cortical Excision, Ann. Surg. **126**: 740 (Nov.) 1947.
5. Penfield, W., and Livingston, S.: Birth Injury, Focal Epilepsy and Cortical Excision: Case Report, Pediatrics **4**: 157 (Aug.) 1949.

develop normally up to the age of 5 months, when he began to have seizures. From this time on there was mental deterioration until the age of 22 months, when surgical removal of an epileptogenic focus was carried out. The cessation of seizures which followed operation has been associated with subsequent general improvement during the seven years which have elapsed since operation.

Radical excision offers hope for many patients who have cortical foci of discharge and whose attacks are not controlled by medication.

PARA-AMINOSALICYLIC ACID IN THE TREATMENT OF PULMONARY TUBERCULOSIS

Bernheim,[1] in 1941, observed that salicylic acid and benzoic acid increase the oxygen consumption and carbon dioxide production of the tubercle bacillus. He concluded that these acids are oxidized as metabolites and that similar chemical configurations possibly play a part in the metabolism of the bacillus. Lehmann,[2] in 1946, investigated a large number of derivatives of benzoic acid and of salicylic acid, searching for a substance with bacteriostatic properties against tubercle bacilli. The most active substance found was 4-aminosalicylic or para-aminosalicylic acid. This substance was bacteriostatic in a concentration of 0.15 mg. per hundred cubic centimeters for the BCG strain of the bovine type of Mycobacterium tuberculosis and exerted a favorable effect on clinical tuberculosis. The inhibitory effect of para-aminosalicylic acid (PAS) in vitro was confirmed by Sievers and by Youmans. In vivo experiments on mice by Youmans and in guinea pigs by Feldman, Karlson and Hinshaw[3] demonstrated the retarding effect of the drug on the development of experimental tuberculosis.

Youmans, Raleigh and Youmans[4] found that para-aminosalicylic acid is highly bacteriostatic in vitro for virulent tubercle bacilli in human infections. Experimental tuberculosis in mice was suppressed by para-aminosalicylic acid when it was administered in the diet in 1 and 2 per cent concentration. Para-aminosalicylic acid and streptomycin when administered to mice simultaneously exerted a suppressive effect on the tuberculous process greater than that of either substance alone. Bloch and his co-workers[5] reported that para-aminosalicylic acid inhibited growth of both the streptomycin-sensitive strain of H-37 RV and the naturally occurring streptomycin-resistant variant, H-37 RVNRI, almost completely in concentration of 1.2 gamma per cubic centimeter. The combination of para-aminosalicylic acid and of streptomycin demonstrated both in culture experiments and in animals a striking inhibitory effect of the combination of the two drugs compared with that of each of the drugs alone. These observations suggest that a combination of the two drugs would permit the use of smaller doses of streptomycin, thus reducing the hazard of toxic effects from the drug and delaying the emergence of streptomycin resistance.

Alin and Difs[6] treated 11 patients at the Serafimer Hospital (Stockholm). These patients had pulmonary lesions sufficiently severe to indicate a thoracoplasty; they were given 14 Gm. of para-aminosalicylic acid daily for one to six months. An equal number of patients with similar lesions served as controls. The only toxic effect noted was an occasional positive Takata reaction and low excretion in the hippuric acid test. The sedimentation reaction during the first month of treatment showed a reduction from 51 to 30 in the treated patients and from 60 to 55 in the untreated. In 3 patients with fever an initial fall of temperature occurred. None of the patients became free from bacilli, nor did any patient escape the indicated thoracoplasty when the general condition allowed the intervention. The only conclusion reached by the Scandinavian investigators was that the investigation should be continued.

Eastlake and Barach[7] treated 12 patients with chronic pulmonary tuberculosis with 10 to 11 Gm. of para-aminosalicylic acid in three to five doses daily for three consecutive weeks with a free interval of one week. One or more courses were given, depending on the tolerance of the patient to the drug. Seven of the seriously ill patients showed a definite decrease in cough, fever and expectoration during the first week. The symptoms recurred after the cessation of treatment. The most consistent side effect of the drug was a diminution in appetite, and some of the patients experienced nausea and vomiting. In 2 patients these symptoms were severe enough to cause cessation of the treatment.

Para-aminosalicylic acid appears to be a therapeutically useful drug in pulmonary tuberculosis, especially in patients from whom streptomycin is withheld for any reason.

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Current Comment

WASHINGTON A. M. A. MEETING—PHYSICIAN TO BE HONORED IN BROADCAST

The clinical program for the Clinical Session of the meeting of the American Medical Association in Washington, December 6-9, will contain many topics of broad appeal. While other aspects of the meeting will be interesting and informative, one of the highlights will be a nationwide broadcast devoted to the life of a physician. This broadcast will be made Wednesday evening, December 7, as a regular part of the "This Is Your Life" series, starring Ralph Edwards. This series is devoted to dramatizing the lives of Americans who have made outstanding contributions to community and national life. One unique feature of this program will be maintained for the December 7 broadcast: The name of the honored guest will not be revealed until the time of the broadcast, and the guest will not know until then that his life will be dramatized. Physicians attending the American Medical Association meeting will be able to attend the broadcast. Complimentary tickets for admission will be made available at the convention.

REPEATED TUBERCULIN TESTS WITHOUT SENSITIZATION

In 1943[1] the demonstration that tubercle bacilli liberate tuberculin (tuberculoprotein) by autolysis led to the discovery of a new tuberculin adapted to transcutaneous tuberculin testing.[2] This tuberculin could be prepared in a finely pulverized hygroscopic form suitable for suspension in a nonirritating, non-water-soluble, but water-pervious, cellulose adhesive. This permitted intimate application to the skin of a highly active but safe material for diagnosis and surveys. Because this test can be used repeatedly and its administration is simple, relation between its frequent use and hypersensitiveness has been questioned. It has been claimed that an Arthus' phenomenon (hypersensitive allergic condition) can develop from the use of large repeated injections of tuberculin. To determine such likelihood, Jones, Nelson and Dwork[3] repeatedly tested 223 children and adolescents in an orphanage. Their ages were 5 to 20 years. Two to four tests were used in each instance. The investigators found that frequent and repeated transcutaneous autolytic tuberculin testing, extending over more than a six month period, did not produce a hypersensitiveness to tuberculin or to the transcutaneous test. They also found that repeated application in this form did not accentuate or suppress an already existing hypersensitiveness to tuberculin.

1. Corper, H. J., and Cohn, M. L.: Autolysis of Tubercle Bacilli and the Production of Tuberculin (Tuberculoprotein), Am. Rev. Tuberc. **48**: 443, 1943.

2. Corper, H. J.; Clark, C., and Nelson, C. R.: Some Features of Autolytic Tuberculin That Adapt it for Special Specific Test Purposes, Tuberculology **8**: 72, 1946.

3. Jones, W. W.; Nelson, C., and Dwork, R. E.: Repeated Transcutaneous Autolytic Tuberculin Tests in Children, Am. Rev. Tuberc. **60**: 45, 1949.

LOCAL RHEUMATIC FEVER PROGRAMS

The New York State Department of Health has issued a "Guide for Local Rheumatic Fever Programs,"[1] developed under the chairmanship of Edward R. Schlesinger, director of the Bureau of Maternal and Child Health. The policies, standards and recommended procedures were examined in detail by the Advisory Committee on Rheumatic Fever to the New York State Department of Health, and numerous suggestions from the committee were incorporated. The Guide seeks to outline the types of children for whom the program is intended, methods for finding them, services they need and means by which these can be provided, and basic standards and procedures for insuring the proper utilization of these services. The program is confined for the time being to services for persons under 21 years of age. A local rheumatic fever program is viewed as a cooperative effort on the part of voluntary and official agencies in the community concerned with the problem and the development of the program as part of a basic public health program in a community with a full time health department. An irreducible minimum for a new program would include adequate follow-up services to insure that a diagnosis is made so far as possible, that recommendations for the care of patients are carried out and that patients are not lost after diagnosis. Direct services required by patients consist of: 1. Medical services for diagnosis and treatment of ambulatory patients. These services should be provided through the family physician, who is the key person in discovering cases of rheumatic fever in its early stages. 2. Consultation services, including laboratory, roentgenologic and electrocardiographic studies. 3. Medical services for the diagnosis and treatment of bed patients at home. 4. Institutional care. 5. Follow-up services, after care and rehabilitation. Vocational guidance and training services should be provided to the patient to prepare him for a useful position in society compatible with his physical disability. Supporting services of the program include: 1. Health education of the general public with the view of disseminating information about rheumatic fever as a public health problem, to arouse public interest and to obtain support for the program. 2. Professional education, to consist of a program of postgraduate education for physicians, nurses and other professional personnel. 3. Case finding and reporting. Special diagnostic and consultation services should be provided to aid the physician in establishing a definite diagnosis. 4. Establishment of a case register as a necessary working tool for the use of the administration in directing follow-up services for continuing care of patients and as an aid in the periodic evaluation of the program to determine its effectiveness. To assist in the organization of local programs, the Bureau of Maternal and Child Health makes available on request the following forms: (*a*) a clinic record for initial medical examination, (*b*) a clinic record for reexaminations, (*c*) a report of initial or changed status of the patient and (*d*) a registration card.

1. Guide for Local Rheumatic Fever Programs, Office of Public Health Education, New York State Department of Health, 1949.

ORGANIZATION SECTION

Official Notes

SECOND ANNUAL PUBLIC RELATIONS CONFERENCE

American Medical Association Headquarters and Bismarck Hotel

PROGRAM

SATURDAY, Nov. 5, 1949

REGISTRATION

9:00 a. m., Auditorium, Ninth Floor, American Medical Association Headquarters.

MORNING SESSION:

9:30 a. m., Auditorium, Ninth Floor, American Medical Association Headquarters.

Address of Welcome.

DR. GEORGE F. LULL, Secretary and General Manager, American Medical Association.

Conference Comments.

LAWRENCE W. REMBER, Public Relations Director, American Medical Association.

ORGANIZING FOR AN OVER-ALL PUBLIC RELATIONS PROGRAM

DR. MAX M. HATTAWAY, Presiding, Chairman, Council on Medical Service and Public Relations, Louisiana State Medical Society.

Organizing the State Public Relations Program at District and County Levels.

DR. DONALD B. KOONCE, Chairman, Public Relations Committee, North Carolina Medical Society.

Working with All Other Committees of the State Society.

DR. PERCY E. HOPKINS, Chairman Committee on Medical Service and Public Relations, Illinois State Medical Society.

Cooperation Desired by Public Relations Committees from State Society and American Medical Association Headquarters.

DR. CHARLES FARRELL, Chairman, Committee on Public Policy and Relations, Rhode Island Medical Society.

Fifteen minute question and answer period after each topic.

LUNCHEON

12:00 noon, Cafeteria, Fifth Floor East, American Medical Association Headquarters.

Those attending are invited to be guests of the American Medical Association for a buffet luncheon in the new cafeteria. Time has been allowed following luncheon for you to tour the new addition to the American Medical Association building.

AFTERNOON SESSION:

1:30 p. m., Auditorium, Ninth Floor, American Medical Association Headquarters.

STATE SOCIETY PUBLIC RELATIONS PROJECTS

DR. A. E. CARDLE, Presiding, Chairman, Health Education Committee, Minnesota State Medical Association.

Tackling the Public Relations Problem of Getting Doctors Into Rural Areas.

DR. J. H. A. PECK, President, Kansas Medical Society.

The PR Value of a Grievance Committee.

DR. GEORGE H. GARRISON, President, Oklahoma State Medical Association.

Press and Radio Relations for County Societies and Individual Doctors.

DR. MCKINNIE PHELPS, Chairman, Public Policy Committee, Colorado State Medical Society.

Full Utilization of the Woman's Auxiliary as a Public Relations Force.

DR. C. ALLEN PAYNE, Chairman, Advisory Committee to the Woman's Auxiliary to the Michigan State Medical Society.

Fifteen minute question and answer period after each topic.

SOCIAL HOUR

5:30 p. m., Green Room, Bismarck Hotel.

DINNER SESSION:

6:30 p. m., Walnut Room, Bismarck Hotel.

DR. LOUIS BAUER, Toastmaster, Chairman, Board of Trustees, American Medical Association.

The Profession and Public Relations.

DR. ERNEST E. IRONS, President, American Medical Association.

Selling Liberty, A Public Relations Project for Every American.

LEONARD E. READ, President, The Foundation for Economic Education, Inc.

SUNDAY, Nov. 6, 1949

MORNING SESSION:

Get It Off Your Chest.

9:30 a. m., Parlor "H." Bismarck Hotel.

DR. F. S. WINSLOW, Presiding, Chairman, Public Relations Committee, New York Medical Society.

"One of Our Best Projects Is—"

Intermission

What We Should Like to Know.

LUNCHEON SESSION:

12:15 p. m., Walnut Room, Bismarck Hotel.

DR. ELMER L. HENDERSON, Toastmaster, President-Elect, American Medical Association.

Public Relations is Our Business.

AVERELL BROUGHTON, President, Public Relations Society of America.

THE SCIENTIFIC EXHIBIT, SAN FRANCISCO SESSION—JUNE 26-30, 1950

The Scientific Exhibit will be one of the features of the Annual Session of the American Medical Association in San Francisco, June 26-30, 1950.

The nineteen sections of the Scientific Assembly will participate with groups of exhibits dealing with the various branches of medicine, as well as the allied medical sciences. Application blanks for space in the Scientific Exhibit are now available and may be obtained from the Director, Scientific Exhibit, American Medical Association, 535 N. Dearborn Street, Chicago 10, Ill.

UNIVERSITY OF MICHIGAN LIBRARIES

Washington Letter

(From a Special Correspondent)

Oct. 24, 1949.

Foreign Military Medical Leaders Greeted in Capital

European and Canadian military medical chiefs were entertained in Washington October 21, en route to the twelfth International Congress of Military Medicine and Pharmacy held in Mexico City October 28-29. Members of the group were Lieut. Gen. Sir Neil Cantlie, director general of British Army Medical Services; Brig. William Coke, Canadian Army medical director; Wing Comdr. J. H. Neal, Royal Air Force; Brig. Georges Apalakis, Greek Army surgeon general; Prof. Sanvenero Rosselli, official delegate from Italy; Major Gen. David Lindsjo, surgeon general of Swedish Defense Forces; Major Gen. Johannes Heinenen, surgeon general of the Finnish Army, and Col. Brig Hans Meuli, Swiss Army surgeon general.

With a planned stopover at the Air Force School of Aviation Medicine, San Antonio, Texas, the military party left Washington for Mexico City October 22. Traveling in the same plane were the following U. S. delegates: Army Surgeon General Raymond W. Bliss, Navy Surgeon General Clifford A. Swanson, Air Force Deputy General Harry G. Armstrong, Major Gen. George E. Armstrong, Rear Adm. Joel T. Boone, Brig. Gen. Wallace H. Graham, Brig. Gen. Edward J. Kendricks and Dr. R. C. Williams, assistant surgeon general of Public Health Service in charge of the Bureau of Medical Services.

State Health Officers Act on Various Issues

The Association of State and Territorial Health Officers, holding its annual meeting here October 19-22, adopted resolutions on a variety of subjects, texts of which will be made public later. Among those who addressed the four day conference were Surgeon General Leonard A. Scheele, Dr. Richard L. Meiling, director of the Office of Medical Services in Department of Defense, and Federal Security Administrator Oscar R. Ewing. The session of Congress that adjourned October 19 "displayed more interest in health measures than any Congress in history," said Ewing. Reviewing its achievements, he cited final enactment of a bill amending the Hill-Burton Act to double—to $150,000,000 annually—the federal contribution toward hospital construction; Senate passage of legislation for support of local public health units, medical schools and school health services, and House committee approval of the medical school assistance bill.

Defense Secretary Johnson Reaffirms Medical Objectives

Secretary of Defense Louis Johnson on October 17 directed a special memorandum to the secretaries of the Army, Navy and Air Force reminding them that "the primary mission of the medical services of each of the military departments is the support of the respective combatant arms and that the organization of the medical services should be scaled to that end."

"Medical training, medical research and the hospital services of the Department of Defense must be constantly directed to the accomplishment of the primary mission as stated above and all nonmilitary activities must, therefore, assume a secondary role," the extraordinary directive stated.

Implicit in the memorandum, according to military spokesmen, was notice that fundamental medical research, postgraduate medical training and other activities which are not translatable rapidly into health benefits for uniformed personnel are to be deemphasized. The action taken by Secretary Johnson was at the request of the Armed Forces Medical Advisory Committee, of which Charles P. Cooper is chairman.

Children's Bureau Engages Five Medical Consultants

Katharine F. Lenroot, chief of the U. S. Children's Bureau, has appointed five prominent authorities in specialized fields of maternal and child health as part time consultants to the Bureau's Division of Health Services. They are Dr. Harry H. Gordon, professor of pediatrics, University of Colorado, and an expert in care of prematurely born infants; Dr. William G. Hardy, director of the Hearing and Speech Center at Johns Hopkins University School of Medicine; Dr. Meyer A. Perlstein of Chicago, who will advise on development of state programs for cerebral palsied children; Dr. Grete L. Bibring, chief of psychiatric services at Beth Israel Hospital, Boston, consultant on mental health aspects of state child health programs, and Dr. John Whitridge Jr. of Baltimore, who will be available for consultation on obstetric services, particularly in rural areas.

"This service marks a new departure in consultation provided by the Children's Bureau," said Dr. Leona Baumgartner, associate chief of the Children's Bureau, "in that it will begin to make available throughout the country top quality medical advice in these fields, heretofore available only in a relatively few states. With these consultants available through the Bureau, more states will be able to improve the quality of care provided in their maternal and child health and crippled children's programs."

Coming Medical Meetings

American Medical Association Clinical Session, Washington, D. C., December 6-9. Dr. George F. Lull, 535 N. Dearborn, St., Chicago, Secretary.

Annual Conference of State Secretaries and Editors, Chicago, A. M. A. Building, Nov. 3-4. Dr. George F Lull, 535 N. Dearborn St., Chicago 10, Secretary.

American Academy of Pediatrics, San Francisco, Nov. 14-17. Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill., Secretary.

American Medical Women's Association, Midyear Meeting, Tucson, Ariz., Nov. 10-11. Dr. Grace Talbott, 49 W. 49th St., New York 20, Corresponding Secretary.

American Society for the Study of Arteriosclerosis, Chicago, Hotel Knickerbocker, Nov. 5-7. Dr. O. J. Pollak, Quincy City Hospital, Quincy 69, Mass., Secretary.

American Society of Anesthesiologists, New York, Hotel New Yorker, Dec. 7-10. Dr. Curtiss B. Hickcox, 188 W. Randolph St., Chicago 1, Secretary.

American Society of Tropical Medicine, Memphis, Tenn., Nov. 6-9. Dr. Frederick J. Brady, National Institute of Health, Bethesda 14, Md., Secretary.

Association of Military Surgeons of the United States, Washington, D. C., Nov. 10-12. Col. James M. Phalen, Army Institute of Pathology, Washington 25, D. C., Secretary.

Central Society for Clinical Research, Chicago, Drake Hotel, Nov. 4-5. Dr. Kenneth G. Kohlstaedt, 960 Locke St., Indianapolis 7, Secretary.

International College of Surgeons, United States Chapter, Atlantic City, Haddon Hall, Nov. 7-12. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago 10, Secretary.

Inter-State Post Graduate Medical Association of North America (International Medical Assembly), Philadelphia, Oct. 31-Nov. 3. Dr. Tom B. Throckmorton, Equitable Bldg., Des Moines, Iowa, Secretary.

Neurosurgical Society of America, Quebec, Canada, Chateau Frontenac, Oct. 30-Nov. 1. Dr. Edward Schlesinger, 710 W. 168th St., New York, Secretary.

Post Graduate Medical Assembly of South Texas, Houston, Nov. 29-Dec. 1. Dr. E. Trowbridge Wolf, 229 Medical Arts Bldg., Houston, Secretary.

Puerto Rico, Medical Association of, San Juan, Dec. 14-18. Dr. Juan Basora-Defillo, 1459 America St., Santurce, Secretary.

Radiological Society of North America, Cleveland, Dec. 4-9. Dr. Donald S. Childs, 713 E. Genesee St., Syracuse 2, N. Y., Secretary.

Southern Medical Association, Cincinnati, Nov. 14-17. Mr. C. P. Loranz, Empire Building, Birmingham, Ala., Secretary.

Southern Psychiatric Association, New Orleans, Nov. 27-29. Dr. Newdigate M. Owensby, 384 Peachtree St., N.E., Atlanta, Georgia, Secretary.

Southern Society of Electroencephalography, New Orleans, Nov. 30. Dr. Samuel C. Little, 2111 Highland Ave., Birmingham 5, Ala., Secretary.

Southern Surgical Association, Hot Springs, Va., The Homestead, Dec. 6-8. Dr. John C. Burch, 2112 West End Ave., Nashville 5, Tenn., Secretary.

Southwestern Medical Association, Albuquerque, New Mexico, Nov. 9-12. Dr. Wickliffe R. Curtis, First National Bank Bldg., El Paso, Texas, Secretary.

Western Surgical Association, Santa Barbara, Calif., Nov.29-Dec. 2. Dr. Warren H. Cole, 1853 W. Polk St., Chicago 12, Secretary.

GOVERNMENT SERVICES

ARMY

PHARMACISTS WANTED

Pharmacists for civilian employment with the Army of Occupation in Japan are urgently needed. These positions pay up to $3,727 per year plus a 10 per cent foreign allowance and free housing. Transportation is furnished by the government. Because of a shortage of suitable housing, dependents are not permitted. Persons selected for these positions must agree to remain a minimum of two years. Pharmacists interested should contact The Department of the Army, Overseas Affairs Branch, 1660 East Hyde Park Boulevard, Chicago.

EXHIBIT OF MEDICAL STAMPS

An exhibit of stamps issued in honor of medicine is on display at the Army Medical Library, Seventh and Independence avenues, Washington, D. C. It is now possible, the announcement states, to provide philatelic material for nearly all the great landmarks in the history of medicine. Stamps of medical and scientific interest have been issued in large numbers in the last twenty years. Expert philatelists and collectors will find the exhibit of particular interest.

OFFICERS CERTIFIED AS SPECIALISTS

Five more Army medical officers have recently been certified as specialists by American boards, according to an announcement by the Surgeon General's office. Four of the new specialists are in the Regular Army and one is in the Medical Corps Reserve. Two were certified in internal medicine and one each in surgery, urology and radiology. The two internists are Lieut. Col. Edward T. Spunt (MC,R) and Major William M. Webb (MC). Col. George L. Beatty (MC) was certified in surgery, Lieut. Col. Romeyn J. Healy Jr. (MC) in radiology and Major John N. Furst (MC) in urology.

COURSES IN RADIOLOGIC DEFENSE

The following three week courses in chemical warfare, radiologic defense and associated subjects will be given at the Chemical Corps School, Edgewood, Md. The courses are designed especially for physicians.

Opening Date	Closing Date	Opening Date	Closing Date
January 9	January 27	May 1	May 19
January 30	February 17	June 5	June 23
March 6	March 24	July 10	July 28
April 3	April 21		

NAVY

NAVY'S HEALTH RATE AT HIGHEST PEAK

Rear Admiral C. A. Swanson, the Surgeon General, reports that the Navy's health rate is higher than at any time since complete medical department records were started in 1850. The incidence rate for diseases, injuries and poisonings among naval personnel dropped during the calendar year 1948 to the lowest ever recorded—442.8 per thousand. The period 1943-1947 was the longest period during which the annual incidence rate for all causes remained below 500 per thousand. A decrease in communicable and noncommunicable diseases among naval personnel is primarily responsible for the improved health rate.

Of the twenty-seven diagnostic classes of diseases, only one reversed the general trend toward lower incidence rates. Class VIII, which includes common infectious diseases of the respiratory type, such as common colds, increased from 100 in 1947 to 104.3 in 1948. The incidence rate for venereal diseases continued its downward trend. The rate for 1948 was 66.6 per thousand, compared with 85.8 in 1947 and 71.6 in 1946. During 1948 the rate declined from 81.9 in January to 50 in December.

The incidence for tuberculosis declined from 1.8 per thousand average strength in 1947 to 1.1 per thousand in 1948. This reduction of about 40 per cent shows the effect of the Navy's intensive campaign to locate and treat infected persons before they become infectious to others. The incidence rate for mental diseases declined slightly, from 8 to 7.6 per thousand, in 1948.

The death rate among naval personnel for all causes in 1948 was minutely higher than the previous year, 2.04 per thousand average strength as compared with 2.03 in 1947. However, the Navy's death rate was lower than in 1948 in only two other years. Although the total death rate has fallen, the share ascribed to injuries has steadily increased. In 1948, injuries were responsible for 81 per cent of all deaths, with a rate of 1.66 per thousand strength, while the rate for disease deaths was only 0.38 per thousand.

CERTIFIED BY SPECIALTY BOARDS

Captains Robert W. Babione, H. K. Sessions and Sterling S. Cook and Comdr. Robert E. Rock have been certified by the American Board of Preventive Medicine and Public Health in the Founders Group.

DUTY UNDER INSTRUCTION

The following medical officers have been nominated for duty under instruction in the Graduate Medical Training Program:

Comdr. William M. Enright, to a fellowship in dermatology and syphilology, Washington University School of Medicine, St. Louis, Missouri.

Comdr. John S. Gruggel, to instruction in industrial medicine, Harvard School of Public Health, Boston.

Comdr. Eugene P. Harris, to a residency in otolaryngology, Naval Hospital, National Naval Medical Center, Bethesda, Md.

Comdr. Herbert Wilson Jr., to a residency in pathology, Naval Hospital, Long Beach, Calif.

Lieut. Comdr. Philip L. Nova, to a residency in surgery, Naval Hospital, San Diego, Calif.

Lieut. Comdr. John D. Boland (MC, USN) to a residency in psychiatry at the Naval Hospital, Philadelphia.

Lieut. Comdr. William A. McCausland (MC, USNR) to instruction in aviation medicine at the Naval School of Aviation Medicine and Research (NAS) Pensacola, Fla.

Lieut. James N. Sussex, to a fellowship in child psychiatry, Philadelphia Child Guidance Clinic, Philadelphia.

Lieut. Charles A. Patterson (MC, USNR) to instruction in aviation medicine at the Naval School of Aviation Medicine and Research (NAS) Pensacola, Fla.

Lieut. (jg) Frank F. Allen, to a residency in surgery, Naval Hospital, Chelsea, Mass.

Lieut. (jg) Frank H. Austin Jr., to a residency in surgery, Naval Hospital, Long Beach, Calif.

Lieut. (jg) Merrill A. Bender, to a residency in radiology, Naval Hospital, National Naval Medical Center, Bethesda, Md.

Lieut. (jg) Larry J. Hines, to a residency in surgery, Naval Hospital, San Diego, Calif.

Lieut. (jg) Cunningham R. MacCordy, to a residency in anesthesiology, Naval Hospital, National Naval Medical Center, Bethesda, Md.

Lieut. (jg) Richard B. Means Jr., to instruction in submarine medicine, Naval Submarine School, Submarine Base, New London, Conn.

Lieut. (jg) Robert F. Neal, to a residency in pediatrics, Naval Hospital, National Naval Medical Center, Bethesda, Md.

Lieut. (jg) Edward E. Parker, to a residency in surgery, Naval Hospital, Great Lakes, Ill.

Lieut. (jg) Harvey O. Randel, to instruction in pediatrics, University of Pennsylvania Graduate School of Medicine, Philadelphia.

Lieut. (jg) Thomas W. D. Smith, to a residency in internal medicine, Naval Hospital, Oakland, Calif.

Lieut. (jg) Harry W. Vandever, to instruction in aviation medicine, Naval School of Aviation Medicine and Research, Naval Air Station, Pensacola, Fla.

Lieut. (jg) Raymond H. Watten, to a residency in internal medicine, Naval Hospital, Long Beach, Calif.

Lieut. (jg) John R. McLaren (MC, USN) to a residency in obstetrics and gynecology at the Naval Hospital, Philadelphia.

Lieut. (jg) Raymond H. Watten (MC, USN) to a residency in internal medicine at the Naval Hospital, Long Beach, Calif.

UNIVERSITY OF MICHIGAN LIBRARIES

AIR FORCE

APPOINT CIVILIAN CONSULTANTS

Twenty civilian physicians, representing various medical specialties, have been named consultants to the U. S. Air Force Medical Service, Major Gen. Malcolm C. Grow, the Surgeon General, has announced. Consultants must be qualified to teach medical officers of the Air Force Medical Service in continuation of their residency training in fulfilment of specialty board requirements; in addition, they advise and counsel the Surgeon General on matters that apply particularly to aviation medicine.

The consultants appointed are:

Dr. Conrad Berens, New York, ophthalmology.

Dr. Paul A. Campbell, Chicago; Dr. Samuel J. Crowe, Baltimore, and Dr. William A. Morgan, Washington, D. C., otolaryngology.

Dr. R. Lee Clark, Houston, Texas, and Dr. Alton Ochsner, New Orleans, surgery.

Dr. Alfred R. Shands Jr., Washington, D. C., orthopedic surgery.

Dr. Robert E. Moran, Washington, D. C., plastic surgery.

Dr. Cortez F. Enloe, Manhasett, ong Island, N. Y.; Dr. Elmer L. Henderson, Louisville, Ky.; Dr. William P. Holbrook, Tucson, Ariz.; Dr. Philip T. Kness, Columbus, Ohio, and Dr. Russell V. Lee, Palo Alto, Calif., internal medicine.

Dr. Herbert B. Wright, Cleveland, urology.

Dr. Frank Fremont-Smith, Massapeque, Long Island, N. Y.; Dr. Donald W. Hastings, Minneapolis, and Dr. John M. Murray, Boston, neuropsychiatry.

Dr. Charles E. Kossman, New York, cardiology.

Dr. W. Randolph Lovelace, Albuquerque, N. Mex., aviation medicine.

Dr. Howard A. Rusk, New York, physical medicine.

OFFICERS TO VISIT MEDICAL COLLEGES

In an effort to spur procurement of medical officers for the U. S. Air Force Medical Service, three teams of officers will visit medical colleges throughout the United States. Each team will be composed of a medical officer engaged in a professional activity, a dental officer, and a medical officer-pilot. Arrangements will be made with deans and professors of military science and tactics of the colleges in advance, so that the greatest numbers of students possible may be personally contacted. One team will have Washington, D. C., as a base of operation, another, Dayton, Ohio, and the third, San Antonio, Texas.

PERSONAL

Lieut. Col. Fratis L. Duff, of the Air Force Medical Service, has been accepted for graduate training at the Johns Hopkins University School of Public Health, Baltimore.

PUBLIC HEALTH SERVICE

REGULAR CORPS EXAMINATION FOR MEDICAL OFFICERS

A competitive examination for appointment of medical officers in the Regular Corps of the United States Public Health Service will be held Jan. 9-11, 1950, at a number of points throughout the United States. Applications must be received no later than Dec. 12, 1949. All commissioned officers are appointed to the general service and are subject to change of station. Appointments will be made in the grades of assistant surgeon (first lieutenant) and senior assistant surgeon (captain). Appointments are permanent and provide opportunities to qualified physicians for a lifetime career in clinical medicine, research and public health.

Requirements for appointment in the grade of assistant surgeon: the applicant must be a citizen of the United States, at least 21 years of age and a graduate from a recognized school of medicine. Physicians who are successful in the examination and are now serving internships will not be placed on active duty in the Regular Corps until completion of internship. Applicants for appointment in the grade of senior assistant surgeon must meet the aforementioned requirements and must have had a total of at least ten years of educational training and professional experience subsequent to high school. The entrance examination will include written professional tests, an oral interview and a physical examination.

Gross pay is governed by the Career Compensation Act of 1949 and is identical to that of officers of equivalent rank in the Army and Navy. Under current law, entrance pay for an assistant surgeon with dependents is $5,686.56 per annum, and for senior assistant surgeon with dependents, $6,546. These figures include the $1,200 annual additional pay received by medical officers as well as subsistence and rental allowance. Retirement pay after thirty years of service or at the age of 64 is three fourths of annual base pay at the time of retirement. Retirement for disability is authorized under the Career Compensation Act and disability retirement pay is at a minimum, one half of the annual base pay at the time of retirement. Additional benefits include thirty days' annual leave, sick leave, full medical care and many of the usual privileges extended to members of the military forces.

Application forms may be obtained from the Surgeon General, United States Public Health Service, Federal Security Agency, Washington 25, D. C.

VETERANS ADMINISTRATION

MORE LIBERAL COMPENSATION RATES

Public Law 339, liberalizing compensation benefits for veterans, becomes effective Dec. 1, 1949. The new law will:

1. Provide additional compensation for veterans with dependents who are rated 50 per cent or more disabled. Heretofore, only veterans with dependents rated 60 per cent or more disabled were eligible to receive additional compensation.

2. Raise the rate on compensation for World War I veterans with "presumed" service-connected disabilities to the rate currently being paid to veterans whose disabilities are determined to be directly service connected.

3. Establish new progressive compensation ratings for veterans with arrested tuberculosis. It provides for 100 per cent rating for the first two years following the date the disease was arrested. For four years after that, the rate is set at 50 per cent. The law also fixes disability ratings for longer periods and requires a veteran to submit to examination and follow prescribed treatment. Failure to do so may result in his rating being reduced from 100 per cent to 50 per cent for the two years following the arrest of the disease.

4. Increase the death compensation to wartime widows with one or more children.

5. Liberalize present rules on compensation for injury or disease incurred while under military or civil confinement. It holds such confinement to be "in line of duty," provided a court-martial sentence of dishonorable discharge is remitted, or in cases of civil confinement the offense does not involve a felony.

For all World War II veterans with service-connected disabilities and those World War I veterans whose disabilities have been determined by the Veterans Administration to be directly service connected, the 8.7 per cent across-the-board increase will apply. For example, a veteran who is 10 per cent disabled and comes under either of those two categories will receive an increase in his monthly compensation check from $13.80 to $15. Others in those categories whose disabilities range up to the 100 per cent maximum will receive proportionate increases in their checks. Thus, such a 100 per cent disabled veteran will get $150 instead of the $138 he now receives. Peacetime rates for service-connected disabilities are fixed by law at 80 per cent of wartime rates. Thus, they will be automatically increased by the new law also.

Medical News

(Physicians will confer a favor by sending for this department items of news of general interest: such as relate to society activities, new hospitals, education and public health. Programs should be received at least two weeks before the date of meeting.)

CALIFORNIA

"Medical Practice in the Changing Order."—A series of six round table discussions on "The Practice of Medicine in the Changing Order" began October 4 and will be presented on the first Tuesday of each month thereafter. The series is sponsored by the School of Public Health and the University of California Medical School and by the Alameda County Medical Association, in cooperation with the University of California Extension. The meetings will be held at 8 p. m. at 159 Forestry Building, Berkeley Campus. The purpose of the series is "a completely informed medical profession capable of exercising continuous leadership in the complex fields of medical economics, prepayment plans, distribution of medical care, public medical care programs, foreign medical care plans and health legislation. A registration fee for all but interns and residents will be charged. Attendance is limited to Doctors of Medicine.

ILLINOIS

Symposium on Industrial Medicine.—A Symposium on Industrial Medicine, which has been arranged by the committees on industrial health of the Chicago Medical Society and the Illinois State Medical Society; the Council on Industrial Health, American Medical Association; the Division of Industrial Health, Illinois Department of Public Health, and the department of preventive medicine and public health, University of Illinois College of Medicine, will be held at the University of Illinois College of Medicine, Chicago, November 2 at 2 p. m. The speakers will be: Drs. George F. Lull, Secretary and General Manager of the American Medical Association, Chicago; William J. Fulton, medical director, General Motors Corporation, Detroit; Joseph H. Chivers, medical director, Crane Company, Chicago; Jean S. Felton, medical director, Oak Ridge National Laboratory, Oak Ridge, Tenn.; Milton H. Kronenberg, assistant medical director, Caterpillar Tractor Company, Peoria; Clarence O. Sappington, industrial health consultant, Chicago, and Leonard F. Weber, clinical professor of dermatology, University of Illinois, Chicago. A dinner will be served at the Graemere Hotel at 6 p. m., after which the meeting will continue. All those interested in industrial medicine are invited to attend.

Chicago

Dr. Van Dellen Appointed Assistant Dean.—Dr. Theodore R. Van Dellen, assistant professor of medicine, Northwestern University Medical School, Chicago, has been appointed assistant dean succeeding Dr. George H. Gardner, who resigned. Dr. Van Dellen received his medical degree in 1936 from Northwestern University Medical School, Chicago. He is director of the Florsheim Heart Clinic at the university and author of the daily health column in the Chicago *Tribune*.

INDIANA

Medical School Scholarships.—The Indiana State Medical Association has awarded medical school scholarships to Kermit Q. Hibner of Indianapolis, a freshman in the Indiana University School of Medicine, and Fred Harless of LaGrange, a sophomore. After completion of their internships, each has agreed to practice in a rural community needing medical services. Scholarships, worth $500 annually, are held by five other medical school students who likewise will enter rural practice in accordance with scholarship requirements.

MASSACHUSETTS

Dr. Stevenson Goes to Pittsburgh.—Dr. Stuart S. Stevenson, formerly assistant professor of child health at Harvard Medical School, Boston, accepted the appointment of research professor of pediatrics at Children's Hospital of Pittsburgh and the University of Pittsburgh School of Medicine effective September 1. Dr. Stevenson is a graduate of Yale University Medical School, 1939. He was on the faculty of Yale University, 1940-1943, before he became associated with Harvard University. He was an assistant surgeon of the U. S. Public Health Service 1943-1946 and served on the staff of the health commission of the Rockefeller Foundation 1944-1946.

MICHIGAN

Lectures on "Frontiers in Chemistry."—The annual "Frontiers in Chemistry" lecture series sponsored by the International Society for the Friends of the Kresge Hooker Library and Wayne University's department of chemistry began on October 10 and will continue through November 28. The programs are scheduled for 7 p. m. in room 314 of Wayne's main building. The remaining lectures are as follows:

Oct. 31, Paul D. Bartlett, Ph.D., Cambridge, Mass., Recent Work on the Mechanism of Peroxide Reactions.

Nov. 7, Lee I. Smith, Ph.D., Minneapolis, Chemistry of Some Cyclopropanes.

Nov. 14, Albert L. Lehninger, Ph.D., Chicago, The Enzymatic Oxidation of Fatty Acids.

Nov. 21, Maurice L. Huggins, Ph.D., Rochester, N. Y., Properties of Long-Chain Compounds.

Nov. 28, D. C. Grahame, Ph.D., Amherst, Mass., Progress in the Study of the Electrical Double Layer.

The noncredit registration fee for the series is $5.

MONTANA

Dr. Larson to Succeed Dr. Parker at Hamilton.—Dr. Carl L. Larson has been appointed director of the Rocky Mountain Laboratory of the Public Health Service at Hamilton to succeed the late Ralph R. Parker, Ph.D. (THE JOURNAL, Oct. 8, 1949, p. 399). Dr. Larson has been assistant chief of the Laboratory of Infectious Diseases, Microbiological Institute of the U. S. Public Health Service. He was born at Butte, Mont., Jan. 28, 1909, received his B.S. from Montana State College, Bozeman, in 1932 and spent six years investigating wildlife disease in Minnesota for the Bureau of Biological Survey of the U. S. Department of Agriculture. He received his M.D. from the University of Minnesota Medical School, 1939.

NEW YORK

Postgraduate Instruction.—The Medical Society of the State of New York in cooperation with the New York State Department of Health has arranged a one day meeting for the Monroe County Medical Society to be held at the Academy of Medicine Building, Rochester, November 10 at 9:45 a. m. The speakers will be:

Stanley E. Bradley, New York, Renal Insufficiency.
J. William Hinton, New York, Upper Gastrointestinal Tract Bleeding.
George G. Deaver, New York, Rehabilitation of the Hemiplegic.
Howard C. Stewart, Albany, BCG.

New York City

Memorial Hospital for Chronic Diseases.—The cornerstone for the new Bird S. Coler Memorial Hospital on Welfare Island was set October 10, with Mayor William O'Dwyer officiating. The 2,000 bed hospital will be the second of three units which will eventually designate Welfare Island as a medical center for the care and treatment in long term cases. The building will contain an auditorium to accommodate 1,300, with a balcony for additional wheelchair patients. The hospital will be affiliated with the New York Medical College, Flower and Fifth Avenue Hospitals, which will provide the professional staff.

Dr. Pitts to Head Department at Cornell.—Dr. Robert F. Pitts, professor of physiology at Syracuse University College of Medicine since 1946, will return to the Cornell University Medical College faculty in New York on July 1, 1950, to become head of the department of physiology and biophysics and professor of physiology. From 1942 to 1946 he was in the physiology department at Cornell University Medical College. Dr. Pitts obtained his Ph.D. degree at Johns Hopkins University, Baltimore, in 1932 and his M.D. degree from New York University in 1938. From 1938 to 1940 he was a fellow of the Rockefeller Foundation. He spent one year at the Neurological Institute at the Northwestern University Medical School and another year at the Eldridge Reeves Johnson Foundation for Medical Physics at the University of Pennsylvania College of Medicine, Philadelphia.

OHIO

Honor 50 Year Practitioners.—Six Trumbull County physicians who have completed 50 years of medical service were honored October 12 by the Trumbull County Medical Society in Youngstown. A certificate and gold pins from the Ohio State Medical Association were presented by Dr. Paul A. Davis, Akron, counselor for the sixth medical district, to Drs. David R. Williams, Girard; John C. Henshaw and Simon S. MacKenzie, Warren; Beman G. McCurley, Cortland, and John M. Elder and Henry V. Ormerod, Niles.

UNIVERSITY OF MICHIGAN LIBRARIES

Lectures on the Lead Problem.—A course for physicians, entitled "The Lead Problem in Industry," will be presented November 7-11 at the Kettering Laboratory, University of Cincinnati College of Medicine. Among the guest speakers on the program will be:

Mr. Robert L. Ziegfeld, New York, Present Industrial Significance of Lead.
Leslie Silverman, Sc.D., Boston, Plant Survey by Engineering Technics.
Elston L. Belknap, Milwaukee, Diagnosis.
Mr. Harry W. Wheeler, Baton Rouge, La., Methods of Control.

Registration, which will be limited to 35 persons, must be made in advance and accompanied with the tuition fee of $50, which may be mailed to Communications, c/o Dr. Wm. F. Ashe, Institute of Industrial Health, College of Medicine, Cincinnati 19. Checks should be made payable to the Kettering Laboratory.

PENNSYLVANIA

Society Election.—The Reading Eye, Ear, Nose and Throat Society at its recent meeting elected the following officers for the coming year: Drs. William J. Hertz, Allentown, president; Roy Deck, Lancaster, president-elect; Philip R. Wiest, Reading, treasurer, and Paul C. Craig, Reading, secretary.

New Research Laboratory.—Ground was broken September 27 for the new $4,000,000 medical research laboratories to be erected by Sharp and Dohme at West Point, Pa., before officials of Montgomery County and civic, church and business leaders. John S. Zinsser, Philadelphia, chairman of the board of Sharp and Dohme, welcomed the visitors and pointed out that the cooperation of the people of the community had made the choice of the new plant location a happy one. Brief addresses were presented by H. Clayton Stauffer, president, Board of Education, Upper Gwynedd Township; T. Malcolm Williams, president, Board of Supervisors, Upper Gwynedd Township, and Frank Martin, president, Lansdale Chamber of Commerce. A luncheon for the official party was held in the plant cafeteria. The four story building, which will contain 110,000 square feet of floor space, is expected to be completed in the spring of 1951.

Pittsburgh

Gibson Laboratory Established.—The University of Pittsburgh School of Medicine has announced the opening of the Gibson Laboratory, which was established by a grant from the Addison H. Gibson Foundation. The laboratory provides additional facilities for studies in applied physiology in the University, carries on investigations of arteriosclerosis and provides service facilities for handling radioactive isotopes in the university. Dr. Campbell Moses Jr. has been appointed director of the laboratory.

TEXAS

Pilot Study on Diabetes.—Dallas City Health Department, the Dallas Diabetic Association and the Dallas County Medical Society are sponsoring a pilot study of diabetes education and control in the city. Dallas is one of four cities in the nation chosen by the U. S. Public Health Service for a study of the disease. They plan to determine how the city's public health facilities can be used in a diabetes program. Dr. James W. Bass, city health officer, will direct the program and will be assisted by members of the U. S. Public Health Service.

New Children's Hospital.—The new Driscoll Foundation Hospital for Children will be erected in Corpus Christi to serve children up to 16 years of age. Funds for the construction and operation of the hospital will be provided from the Driscoll Foundation, established by the late Mrs. Clara Driscoll. The building is designed to accommodate 100 patients in the first project and will be expanded to 200 beds in the future. Facilities include a school classroom for children obliged to remain in the hospital for any appreciable time, and recreational facilities.

WISCONSIN

Loevenhart Memorial Lecture.—The annual Loevenhart Memorial Lecture sponsored by Phi Delta Epsilon Medical Fraternity will be delivered by Dr. Paul D. White of Boston in the University of Wisconsin Medical School, Madison, Auditorium S.M.I., on November 28 at 8 p. m.

Visiting Lecturer from Norway.—Professor Johann Holtz of the University of Norway in Oslo will be at the University of Wisconsin Medical School as a visiting lecturer in the department of surgery. Dr. Holtz' program includes the following addresses: November 8, at 8 p. m., he will address the Medical School Society on "Gastric Carcinoma from a Surgical Point of View." On November 10, at 4 p. m., he will address a convocation of medical students and faculty on "Surgical Treatment of Pulmonary Tuberculosis."

Grant to Aid Pediatric Teaching.—With the aid of a $10,000 grant awarded to the Marquette University School of Medicine, Milwaukee, by the Edward Uhrig Foundation, under the terms of the will of the late Rosa Uhrig of Milwaukee, Marquette University will expand its teaching program in pediatrics by adding a full time physician instructor to its staff. The appointee will carry on teaching activities at the Milwaukee Children's Hospital.

GENERAL

Establish Brain Registry.—The American Academy for Cerebral Palsy has established a brain registry at the Chicago State Hospital. The academy welcomes the receipt of brains of specimens derived from patients who had cerebral palsy and of whom good clinical histories have been taken. Correspondence on this subject should be directed to Meyer A. Perlstein, M.D., 4743 North Drake Avenue, Chicago 25.

Medicolegal Congress.—The Academy of Forensic Sciences will hold its second meeting in Lincoln Hall, Northwestern University School of Law, Chicago, Jan. 26-28, 1950. The meeting will be devoted to a discussion of forensic science problems and a formal organizational program. Persons interested in presenting papers should contact Dr. Abraham W. Freireich, 180 Hempstead Avenue, Malverne, N. Y., or Ralph F. Turner, acting secretary, Department of Police Administration, Michigan State College, East Lansing, Mich.

Dunham Lectureship.—The annual Edward K. Dunham Lectureship for the Promotion of Medical Sciences will be held in the Amphitheater, Building D, Harvard Medical School, Boston, on November 3, 8 and 10 at 5 p. m. Dr. Christopher Howard Andrewes, member of the scientific staff at the National Institute for Medical Research, Hampstead, London, will deliver the three lectures on "Some Properties of Viruses," "Epidemic Influenza" and "The Puzzle of the Common Cold."

Association of Blood Banks.—The second annual meeting of the American Association of Blood Banks will be held in the Olympic Hotel, Seattle, November 3-5, under the presidency of Dr. Ralph G. Stillman, New York. Invited speakers include:

Mrs. Bettina B. Carter, Pittsburgh, Progress in Preparation and Use of Rh Hapten.
James J. Griffitts, Miami, Florida, The "T" Agglutinin Phenomenon in Human and Animal Blood.
Hugh W. Jones, Seattle, Serologic Changes in Lupus Erythematosus Disseminatus.
Ernest E. Muirhead, Dallas, Texas, Incompatible Blood Transfusions: Further Experience with Their Management.
Joseph M. Hill, Dallas, Texas, Antibodies of Different Reactivities.
Emile E. Palmquist, Seattle, How the Health Officer Looks at the Blood Bank.

Meetings on Ophthalmology.—The National Society for the Prevention of Blindness will hold a five day conference in conjunction with the Interim Session of the Pan-American Association of Ophthalmology, March 26-30, 1950, at the Floridian Hotel, Miami Beach, Fla. The theme of the meeting will be "The Americas Unite to Save Sight"; among the subjects to be discussed are current blindness prevention programs in countries of the Western Hemisphere; trachoma; industrial ophthalmology; eye problems of school children, and medical and social management of the glaucomas. Details may be obtained from the National Society for the Prevention of Blindness, 1790 Broadway, New York 19. Reservations should be made with the Floridian Hotel, 540 West Avenue, Miami Beach.

Airline Increases Medical Facilities.—Complete medical facilities for the more than 3,000 Trans World Airline employees in the Atlantic Region have been established at New York's La Guardia Field through consolidation of the airline's international and domestic operations divisions. Eleven separate rooms comprising the medical unit planned by Dr. Harold B. Dye, manager of medical services for the airline, include an audio room, x-ray room, examining room, laboratory and emergency clinic as well as office space for the physicians, nurses and secretarial staff. All flying personnel are examined each year with the exception of hostesses who get a biannual physical examination. Eight persons, including five nurses, a technologist and two secretaries, comprise the present staff under the direction of Dr. Robert Rehm, Wilmington, Del., former Army air flight surgeon, and regional supervisor of medical services for TWA. Two more physicians are to be appointed to the staff.

Cancer Research Fellowships.—An exchange fellowship program whereby American investigators in fundamental cancer research and clinical investigation will study on a fellowship basis in Great Britain, and an equal number of British scientists will be trained at research centers here, has evolved from a visit of representatives of the British Empire Cancer Campaign to the offices of the American Cancer Society. The British-

American Exchange Fellowships in Cancer Research, awarded by the American Cancer Society on the recommendation of the Committee on Growth of the National Research Council, are now open to citizens of the United States who have M.D., S.D. or Ph.D. degrees. Fellowships are awarded for one year and carry a stipend of $4,020 and an allowance of $600 for travel to the place of the fellowship in Great Britain. Application forms may be secured and submitted to the Executive Secretary, Committee on Growth, Division of Medical Sciences, National Research Council, 2101 Constitution Avenue, Washington 25, D. C.

Symposium at Pasadena on Air Pollution.—The Stanford Research Institute in cooperation with the California Institute of Technology, Pasadena; University of California, Berkeley, and the University of Southern California, Los Angeles, is sponsoring a National Air Pollution Symposium at the Huntington Hotel, Pasadena, November 10-11. The four sessions of the symposium will be on the subjects: "Technics of Sampling and Analysis," "Sources and Control of Air-Borne Matter," "Meteorologic Influences and Visibility" and "Biologic Effects of Air-Borne Matter." Louis C. McCabe, Ph.D., chief, Office of Air and Stream Pollution Prevention Research, U. S. Department of the Interior, Bureau of Mines, Washington, D. C., will speak on "National Trends in Air Pollution" at the luncheon on November 10, and W. B. Gibson, chairman, Business and Industrial Economics Department, Stanford Research Institute, will speak on "The Economics of Air Pollution" at the luncheon on November 11.

Southern Medical Association Meeting.—The Southern Medical Association will hold its forty-third annual meeting in Cincinnati November 14-17, under the presidency of Dr. Oscar B. Hunter, Washington, D. C. The Campbell-Kenton County Medical Society of Kentucky will be the host society. General headquarters will be at the Netherland Plaza Hotel. The scientific program will include: general sessions, clinical sessions, section meetings, scientific exhibits and medical motion pictures. Among many other speakers, from various parts of the nation, will be the following officers of the American Medical Association: Dr. Ernest E. Irons, Chicago, president, will address the general session Wednesday evening on "The Deception of Nationalized Medicine," and Dr. Elmer L. Henderson, Louisville, Ky., president-elect, will discuss "Management of the Acute Gallbladder" before the general session Monday afternoon. The motion picture "They Also Serve," prepared by the American Medical Association, will be presented. The program of entertainment includes the president's reception and a golf tournament at the Fort Mitchell Country Club, Fort Mitchell, Ky., and the annual trap and skeet-shooting tournament at the Ryland County Club, Covington, Ky. Various other medical associations will hold their annual meeting during this period.

Program to Produce Psychiatric Films.—At a luncheon meeting of the New York Film Council, October 13, Dr. George Stevenson, president, American Psychiatric Association, announced a program involving an initial expenditure of $250,000 to give the public authoritative films dealing with psychiatry and related subjects. The program will be directed by the Mental Health Film Board, whose newly appointed members are:

Dr. Kenneth Appel, chief, Neuro-Psychiatric Clinic, Pennsylvania Hospital.
Dr. Leo H. Bartemeier, president, International Psychoanalytic Society.
Dr. Leona Baumgartner, Children's Bureau, Federal Security Agency.
Dr. Carl Binger, editor, *Psychosomatic Medicine.*
Dr. Joseph M. Bobbitt, clinical psychologist, National Institute of Mental Health.
Dr. M. Ralph Kaufman, president, American Psychoanalytic Association.
Dr. Thomas A. C. Rennie, associate professor of psychiatry, Cornell University Medical College.
Dr. Howard P. Rome, Mayo Clinic.
Dr. Leon Saul, head, Department of Preventive Psychiatry, University of Pennsylvania School of Medicine.

Dr. Stevenson said that the film board was developed to meet the needs for effective materials giving the public insight into the emotional problems of everyday life. The film program was made possible by the cooperation of leaders in mental health in thirteen states. Mr. Robert Anderson, who produced the psychiatric films "Feeling of Rejection" and "Feeling of Hostility," for the Canadian National Film Board, will be the film officer of the new board. Mr. Anderson said, "It is hoped that seven films will be in production before Christmas and that they will be available for theatrical and nontheatrical distribution by spring. Tentative subjects which are being explored include Mental Health of the Aging, Mental Health in Education, Child Guidance Clinics, and Parent-Child Relationships." The thirteen states participating in the plan are: California, Connecticut, Delaware, Illinois, Iowa, Maryland, Michigan, Nevada, North Carolina, Oklahoma, Puerto Rico, Rhode Island and South Carolina.

Prevalence of Poliomyelitis.—Reports of cases of poliomyelitis for the period indicated has been received from the National Office of Vital Statistics, U. S. Public Health Service.

Division and State	Week Ended Oct. 15, 1949	Week Ended Oct. 16, 1948	Median, 1944-1948	Jan. 1 to Oct. 15, 1949	Jan. 1 to Oct. 15, 1948
New England States					
Maine	8	1	2	419	37
New Hampshire	..	..	1	177	18
Vermont	10	..	1	120	20
Massachusetts	50	9	27	1,609	171
Rhode Island	12	..	1	140	7
Connecticut	19	5	8	524	102
Middle Atlantic States					
New York	182	66	68	4,782	1,211
New Jersey	64	42	26	1,240	658
Pennsylvania	33	29	29	650	611
East North Central States					
Ohio	47	38	38	1,552	1,008
Indiana	43	12	12	947	310
Illinois	79	74	48	2,589	951
Michigan	81†	24	24	2,456†	571
Wisconsin	26	57	49	904	460
West North Central States					
Minnesota	23	79	25	1,676	1,026
Iowa	37	61	34	1,006	896
Missouri	27	21	17	1,203	276
North Dakota	7	4	2	435	110
South Dakota	10	66	2	333	347
Nebraska	21	50	10	545	630
Kansas	24	10	5	645	271
South Atlantic States					
Delaware	..	1	1	38	123
Maryland	15	10	7	199	89
District of Columbia	7	7	6	89	112
Virginia	11	22	12	246	491
West Virginia	5	4	4	315	139
North Carolina	8†	43	10	207†	2,331
South Carolina	3	8	5	97	328
Georgia	8†	4	2	183†	201
Florida	5	8	8	217	231
East South Central States					
Kentucky	21	7	7	555	179
Tennessee	12	12	8	464	331
Alabama	9	6	1	221	189
Mississippi	6	1	1	302	140
West South Central States					
Arkansas	9†	4	4	903†	130
Louisiana	4	8	..	208	133
Oklahoma	29	3	3	1,162	333
Texas	36	32	20	2,631	1,552
Mountain States					
Montana	4	5	5	80	60
Idaho	16	1	3	412	98
Wyoming	..	..	1	101	68
Colorado	31	6	3	582	102
New Mexico	16	4	2	178	69
Arizona	..	5	3	141	123
Utah	19	2	2	215	76
Nevada	2	2	..	15	6
Pacific States					
Washington	21	26	14	506	259
Oregon	33	9*	8	226	140*
California	74	234	30	1,989	3,746
Total	1,207†	1,122*	...	35,943†	21,510*
Median, 1944-1948	....	.....	711	16,134	

* Current corrections made in cumulative totals.
† Deductions: Michigan, 1 case week ended September 3 and 2 cases week ended October 7; North Carolina, 1 case week ended October 1; Georgia, 1 case week ended September 10; Arkansas, 1 case week ended August 6.

CORRECTION

Dosage of Morphine.—In the paper entitled "Acute Craniocerebral Trauma" in THE JOURNAL, Oct. 8, 1949, page 373, the sentence beginning in the ninth line from the bottom of the left hand column should read: "In addition to these physical measures small doses of morphine sulfate (1/8 to 1/12 grain [8 to 5 mg.]) given every two hours are sometimes effective in lowering high temperatures."

Marriages

ANDREW B. STUART, Central Islip, N. Y., to Miss Irene Spanos of Rockville Centre, recently.

WILLIAM COLBERT ADAMSON, Philadelphia, to Miss Katherine King of Bristol, Tenn., August 5.

FRANK M. HEIFETZ, Lowell, Mass., to Miss Shirley I. Newman of Brookline, August 7.

HARRY H. HADDON Jr., to Miss Mary Elizabeth Duck, both of Philadelphia, July 23.

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Deaths

Rosco Genung Leland, formerly director of the Bureau of Medical Economics of the American Medical Association, died at his home in Chicago, October 17, aged 64, of acute coronary occlusion. Dr. Leland was born near Mendon, Mich., June 17, 1885. After his preliminary education at Kalamazoo College and the University of Michigan, he received an M.D. degree from the University of Michigan Department of Medicine and Surgery, in 1909. He became assistant in obstetrics and gynecology at his alma mater. He practiced medicine in East Leroy, Mich., from 1910 to 1914, and at Kalamazoo, where he served also as health officer. During World War I he went overseas with the thirty-second division, C. O., Camp Hospital, 50, Tonnerre, France. On returning to the United States in May 1919, after having received two promotions in France, he remained in the service as surgeon to the First R.O.T.C. Infantry Camp, Camp Custer, Mich. After returning to civil life, Dr. Leland joined the Ohio State Health Department, serving as chief of the division of hygiene and later became executive secretary of the Toledo Public Health Association. Dr. Leland was invited to join the staff of the American Medical Association as assistant director of the Bureau of Health and Public Instruction. In 1931 he became director of the newly formed Bureau of Medical Economics. In 1940 he was directed to assume charge of a medical preparedness study involving the nature, location and quantity of medical personnel in the event of war. During World War II he was appointed a supervisor of the consultant office in the headquarters of the American Medical Association for Procurement and Assignment Service for Physicians, Dentists and Veterinarians and a member of the Health and Medical Committee of the Selective Service System. Dr. Leland was a fellow of the American Public Health Association, a member of the Royal Economic Society of England, the American Economic, American Statistical and National Education associations, Ohio State Medical Association and the American Medical Association.

Ernest Edward Shaw ✠ Indianola, Iowa; born in Mitchellville, Iowa, Aug. 4, 1893; State University of Iowa College of Medicine, Iowa City, 1920; in 1949 member of the House of Delegates of the American Medical Association; past president of the Warren County Medical Society; president of the Iowa section of the American Academy of General Practitioners; formerly vice president of the Iowa Clinical Medical Society; member of the Iowa Heart Society; served during World War II; member of the city council; affiliated with the Iowa Methodist, Iowa Lutheran and Mercy hospitals in Des Moines; member of the board of trustees of the Simpson College; on the editorial board of the *Journal of the Iowa State Medical Society;* died August 27, aged 56, of coronary thrombosis.

Russell Brooks Bailey ✠ Wheeling, W. Va.; born in Weston, W. Va., Oct. 17, 1898; University of Cincinnati College of Medicine, 1922; recently elected president of the West Virginia State Medical Association to assume duties Jan. 1, 1950; member of the council of the West Virginia State Medical Association; president of the West Virginia Cancer Society, director of the American Cancer Society and chairman of the state board of health; specialist certified by the American Board of Surgery; member of the Southeastern Surgical Association; fellow of the American College of Surgeons; affiliated with Ohio Valley General Hospital in Wheeling and Reynolds Memorial Hospital, Glendale; died September 15, aged 51, of coronary occlusion.

Donald Charles Tulloch, Albany, N. Y.; born in Syracuse, Dec. 11, 1907; Syracuse University College of Medicine, 1931; M. P. H., Johns Hopkins University School of Hygiene and Public Health, 1945; assistant district state health officer, Albany District, 1945-1946; district state health officer, New York State Department of Health since 1946; part time local health officer of the town of Smithfield, from 1935 to 1944; instructor in preventive medicine and public health, Albany Medical College; member of the American Medical Association; served as president of the New York State Epidemiological Society; died in South Bethlehem, August 28, aged 41, of a cerebral tumor.

Thomas Krapfel Lewis ✠ Merchantville, N. J.; born in Merchantville, N. J., Jan. 7, 1887; University of Pennsylvania School of Medicine, Philadelphia, 1913; member of the House of Delegates of the American Medical Association from 1941 to 1949; past president of the Medical Society of New Jersey, Camden County Medical Society and the Camden City Medical Society; fellow of the American College of Physicians; served overseas during World War I; affiliated with the Cooper Hospital in Camden; died in New Haven (Conn.) Hospital August 28, aged 62, of coronary infarction.

John Joseph Larkin Jr., Boston; Harvard Medical School, Boston, 1939; member of the American Medical Association and the College of American Pathologists; specialist certified by the American Board of Pathology; assistant professor of pathology at Tufts College Medical School; served in World War II from 1942 to 1945 as captain, later major, at Base Hospital 136 in England and France as chief of the pathologic laboratory; pathologist at the Holy Ghost Hospital in Cambridge, where he was in charge of cancer research, and St. Elizabeth's Hospital; died in Sterling, Mass., August 26, aged 35, of accidental drowning.

Catherine Strunk Amatruda, New Haven, Conn.; born in Ithaca, N. Y., March 4, 1903; Cornell University Medical College, New York, 1926; certified by the National Board of Medical Examiners; specialist certified by the American Board of Pediatrics; member of the American Academy of Pediatrics; assistant professor of pediatrics (child study center), Yale University School of Medicine; associate pediatrician at the Grace-New Haven Community Hospital University Service; died in New Haven Hospital September 1, aged 46, of cerebral hemorrhage.

Jaques Cortelyou Rushmore, Stormville, N. Y.; Long Island College Hospital, Brooklyn, 1903; professor emeritus of clinical orthopedic surgery at his alma mater; member of the American Medical Association; fellow of the American College of Surgeons; served overseas during World War I; consultant on orthopedics at the Veterans Administration Hospital in Castle Point, Brooklyn Hospital, St. John's Hospital, Kings County Hospital and House of St. Giles the Cripple, all in Brooklyn; died September 3, aged 73, of heart disease.

Gonzalo Francis Alcazar, Hartford, Conn.; Universidad de la Habana Facultad de Medicina y Farmacia, Cuba, 1943; resident at the Hartford Hospital; died July 7, aged 29.

Jesse Peck Baird, Dyersburg, Tenn.; Homeopathic Medical College of Missouri, St. Louis, 1903; Barnes Medical College, St. Louis, 1904; member of the American Medical Association; fellow of the American College of Surgeons; affiliated with the Baird-Brewer General Hospital; died July 6, aged 72.

Oskar Leonard Bertelson ✠ Crookston, Minn.; University of Minnesota College of Homeopathic Medicine and Surgery, Minneapolis, 1900; an Associate Fellow of the American Medical Association; division surgeon for the Great Northern Railway; died July 14, aged 70, of uremia.

Hugh Franklin Bowers, Belleville, Ill.; St. Louis University School of Medicine, 1941; member of the American Medical Association; secretary treasurer of the Kaskaskia branch of the American Academy of General Practitioners; affiliated with St. Elizabeth's Hospital; died August 18, aged 33, of coronary thrombosis.

Arthur Curtis Branyon ✠ Fayette, Ala.; Memphis (Tenn.) Hospital Medical College, 1903; consultant, McNease and Robertson Hospital; died August 24, aged 66, of carcinoma of the prostate with metastases to the bones.

Arnold Isaiah Bricker, Brooklyn; Long Island College Hospital, Brooklyn, 1947; certified by the National Board of Medical Examiners; resident at the Cumberland Hospital; died in August, aged 24.

Elmer H. Brubaker, Indianapolis; Physio-Medical College of Indiana, Indianapolis, 1896; member of the American Medical Association; formerly on the staff of the Methodist Hospital; at one time practiced in New Castle, Ind., where he was secretary of the Henry County Board of Health and county health officer; died August 22, aged 80, of congestive heart failure.

John Howard Bryant, North Hollywood, Calif.; Northwestern University Medical School, Chicago, 1903; for many years practiced in Galesburg, Ill., where he was on the staff of Galesburg Cottage Hospital; died August 13, aged 73, of coronary thrombosis.

Benjamin Oscar Burkey ✠ Baltic, Ohio; University of Wooster Medical Department, Cleveland, 1911; served on the staff of the Union Hospital in Dover; died August 21, aged 64, of pulmonary edema.

Arthur Maitland Chatham ✠ Cleveland; Western Reserve University Medical Department, Cleveland, 1905; affiliated with St. Alexis and St. Luke's hospitals; for many years active in Boy Scout work; died August 10, aged 71, of myocardial infarction.

✠ Indicates "Fellow" of the American Medical Association.

Elmina Farquhar Cook, Los Angeles; Drake University Medical Department, Des Moines, 1888; at one time a medical missionary in Palestine; died August 6, aged 89, of carcinoma of the sigmoid colon.

John Jesse Cooper, Cedartown, Ga.; Tennessee Medical College, Knoxville, 1893; member of the American Medical Association; served as councilman and mayor; many times elected to the state legislature as representative and senator; died in the Polk General Hospital August 5, aged 81, of heart failure.

Arthur Donald Cowdrick, Clearfield, Pa.; Medico-Chirurgical College of Philadelphia, 1909; member of the American Medical Association; affiliated with the Clearfield Hospital; died August 19, aged 62, of uremia.

Clement Francis Derezinski ✦ Muskegon, Mich.; Loyola University School of Medicine, Chicago, 1937; lieutenant (jg) in the U. S. Navy during World War II; affiliated with the Mercy and Hackley hospitals; died August 19, aged 37, of bulbar poliomyelitis.

August Doerr ✦ Milwaukee; College of Physicians and Surgeons of Chicago, 1896; affiliated with the Deaconess Hospital, where he died August 6, aged 77, of tabes dorsalis and cerebral hemorrhage.

James Lee Doughton, Sparta, N. C.; College of Physicians and Surgeons, Baltimore, 1893; died August 23, aged 79.

Talbot S. Duff, Cainsville, Mo.; Kansas City (Mo.) Medical College, 1899; member of the American Medical Association; served as health officer; died August 16, aged 71, of coronary disease.

William Taylor Elmore, Gainesville, Fla.; University of the South Medical Department, Sewanee, Tenn., 1904; member of the American Medical Association; served during World War I; died in the Veterans Administration Hospital, Lake City, August 7, aged 76, of arteriosclerosis and cerebral hemorrhage.

Archie Lewis Erwin, Nashville, Tenn.; University of Nashville Medical Department, 1905; member of the American Medical Association; served during World War I; affiliated with the Nashville General Hospital; died in Brentwood August 6, aged 67.

Willard Joshua Fenton, St. Petersburg, Fla.; Keokuk (Ia.) Medical College, College of Physicians and Surgeons, 1904; served during World War I; died August 21, aged 68, of heart disease.

John Winston Fowlkes ✦ New York; University of Virginia Department of Medicine, Charlottesville, 1915; specialist certified by the American Board of Otolaryngology; served on the faculty of the New York University College of Medicine; member of the American Academy of Ophthalmology and Otolaryngology; fellow of the American College of Surgeons; affiliated with St. Luke's and Bellevue hospitals; died in Nantucket, Mass., August 9, aged 58, of coronary thrombosis and hypertension.

William Joseph Francis, East Rockaway, N. Y.; Cleveland Homeopathic Medical College, 1904; specialist certified by the American Board of Radiology; served during World War I; died August 7, aged 71.

Milton Blythe Galloway ✦ Webster City, Iowa; Rush Medical College, Chicago, 1911; member of the American Society of Anesthetists; served as county coroner; died August 9, aged 68, of arteriosclerotic myocarditis.

Arthur Howell Gerhard, Philadelphia; University of Pennsylvania School of Medicine, Philadelphia, 1911; served overseas during World War I; formerly on the faculty of his alma mater; affiliated with the Pennsylvania, Episcopal and University hospitals and the Bryn Mawr (Pa.) Hospital; died August 29, aged 72.

Lawrence Gary Gould, Lombard, Ill.; Chicago Medical School, 1941; member of the American Medical Association; served on the staff of the Memorial Hospital, Elmhurst; died in Idaho Springs, Colo., in August, aged 36.

Charles Oscar Grimes, Sigourney, Iowa; Keokuk (Ia.) Medical College, 1895; died in St. Joseph's Hospital, Ottumwa, August 21, aged 76.

Meyer Hantman ✦ Cleveland; Western Reserve University School of Medicine, Cleveland, 1931; specialist certified by the American Board of Dermatology and Syphilology; served overseas during World War II; assistant clinician, division of medicine at the Mount Sinai Hospital, where he died August 26, aged 41, of subarachnoid hemorrhage and congenital aneurysm of circle of Willis.

William Ralph Harder ✦ Los Gatos, Calif.; University of California Medical School, San Francisco, 1928; member of the American Association of Industrial Physicians and Surgeons; affiliated with O'Connor Hospital, San Jose; died August 16, aged 50, of bilateral bronchopneumonia and acute gangrenous cholecystitis.

Louis Morris Harris ✦ Columbus, Ohio; Ohio State University College of Medicine, Columbus, 1912; formerly county coroner; died August 31, aged 58.

John Franklin Henderson, Oakland, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1911; member of the American Medical Association; served during World War I; died in Paris August 25, aged 68, of coronary occlusion.

Albert Edward Henwood, Mattawan, Mich.; Detroit Homeopathic College, 1905; served during World War I; died August 23, aged 71, of acute coronary occlusion.

Philip Launcelot Hill, Petersburg, Va.; Medical College of Virginia, Richmond, 1917; member of the American Medical Association; formerly a medical missionary in Korea; member of the city council and health officer of Colonial Heights; served as coroner of Chesterfield County and Colonial Heights; died in Petersburg Hospital August 8, aged 60, of cerebral hemorrhage.

Walter Bradley Hill, Long Beach, Calif.; University of Southern California Medical Department, Los Angeles, 1909; affiliated with the Seaside Memorial Hospital; died July 19, aged 74, of coronary thrombosis.

Arthur Fenwick Holding ✦ Albany, N. Y.; Albany Medical College, 1901; specialist certified by the American Board of Radiology; member of the American Roentgen Ray Society and the American Radium Society; at one time on the faculty of Cornell University Medical College in New York and lecturer in roentgenology at his alma mater; served overseas during World War I; affiliated with the Memorial Hospital; died August 27, aged 72, of carcinoma of the esophagus.

Luther Talmadge Hollis ✦ Kansas City, Mo.; University Medical College of Kansas City, 1896; an Associate Fellow of the American Medical Association; died in St. Joseph Hospital August 8, aged 77, of arteriosclerotic heart disease.

Ralph Holt, Paducah, Ky.; Barnes Medical College, St. Louis, 1899; for many years county physician; died in the Riverside Hospital August 11, aged 76, of hypertrophy of the prostate, cerebral vascular disease and pneumonia.

Samuel Reeder Huff ✦ Eldred, Pa.; Medico-Chirurgical College of Philadelphia, 1908; died in the Bradford (Pa.) Hospital August 29, aged 65.

Eugene B. Hultz, Hill City, S. D.; Beaumont Hospital Medical College, St. Louis, 1900; St. Louis University School of Medicine, 1904; member of the American Medical Association; died July 2, aged 71, of cerebral hemorrhage.

Norman William Ingalls, Cleveland Heights, Ohio; Western Reserve University Medical Department, Cleveland, 1905; formerly associate professor of anatomy at his alma mater; member of the American Association of Anatomists; died July 27, aged 68, of coronary thrombosis.

Thomas Arthur Johnson ✦ Rockford, Ill.; Rush Medical College, Chicago, 1911; certified by the National Board of Medical Examiners; member of the International College of Surgeons; fellow of the American College of Surgeons; affiliated with the Swedish American Hospital, where he died August 7, aged 63, of coronary thrombosis.

Karl Esterly Jones Jr., Pompeii, Mich.; Northwestern University Medical School, Chicago, 1948; interned at the Cook County Hospital in Chicago; died in August, aged 27, of poliomyelitis.

Armand George Kersten, Detroit; Detroit College of Medicine and Surgery, 1914; member of the American Medical Association and the American Urological Association; fellow of the American College of Surgeons; served during World War I; affiliated with St. Mary's Hospital, where he died August 30, aged 58, of carcinoma of the colon.

Leon T. Leach, Indianapolis; Medical College of Indiana, Indianapolis, 1901; died in St. Vincent's Hospital August 15, aged 73, of essential hypertension and cerebral hemorrhage.

Howard Taylor Lewis ✦ New York; Columbia University College of Physicians and Surgeons, New York, 1906; consulting surgeon at Lutheran Hospital, where he died August 24, aged 69.

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Frank Levinson, Baltimore; University of Maryland School of Medicine, Baltimore, 1911; died in the Church Home and Hospital recently, aged 59, of cerebral hemorrhage and arteriosclerosis.

Nelson Hiram Lewis, Benkelman, Neb.; National Medical University, Chicago, 1901; Milwaukee Medical College, 1905; died July 21, aged 80, of cerebral hemorrhage.

Alonzo Conduit Lillard, St. Joseph, Mo.; University of Missouri School of Medicine, Columbia, 1898; died August 22, aged 75, of coronary occlusion.

Young Cleveland Lott ✦ Miami, Fla.; University of Louisville (Ky.) Medical Department, 1912; member of the National Gastroenterological Association; served during World Wars I and II; formerly on the staff of the Jackson Memorial Hospital; died August 22, aged 60, of nephrosclerosis with uremia.

Willis Austin McCall ✦ Butler, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1904; served during World War I; died in the Jameson Memorial Hospital, New Castle, July 27, aged 70, of complications following a fall.

William E. MacLaughlin, Glendale, Calif.; University of Minnesota College of Medicine and Surgery, Minneapolis, 1896; member of the American Medical Association; served during World War I; died August 14, aged 77, of bronchogenic carcinoma of the right lung.

George Erland Malmgren ✦ Los Angeles; University of Minnesota Medical School, Minneapolis, 1927; at one time affiliated with the Mayo Foundation in Rochester, Minn.; on the staffs of the Hospital of the Good Samaritan and the Cedars of Lebanon Hospital, where he died August 24, aged 47, of spasmodic torticollis and respiratory failure.

William Judson Martin ✦ Kokomo, Ind.; St. Louis College of Physicians and Surgeons, 1903; past president and secretary of the Howard County Medical Society; served as city and county health officer; past president of the Indiana Tuberculosis Association and the Howard County Tuberculosis Association, which he had founded; died in St. Joseph Hospital August 24, aged 79, of cerebral hemorrhage.

Laurent Obid Miller, Grand Chenier, La.; Medical Department of Tulane University of Louisiana, New Orleans, 1908; died August 18, aged 68, of heart failure.

Morrill Justin Morris, Seattle; Jefferson Medical College of Philadelphia, 1915; member of the American Medical Association; member of the Pacific Coast Oto-Ophthalmological Society; served during World War I; died in Salina, Utah, August 25, aged 58.

Walter Franklin Mylin ✦ Willow Street, Pa.; Jefferson Medical College of Philadelphia, 1903; died August 2, aged 71, of carcinoma of the prostate.

A. Victor Nasatir ✦ Los Angeles; University of Illinois College of Medicine, Chicago, 1924; fellow of the American Public Health Association; member of the American Association of Industrial Physicians and Surgeons; director of the industrial hygiene division of the city health department; served during World War II; formerly affiliated with the Cedars of Lebanon Hospital and the Los Angeles County General Hospital; at one time lecturer at the College of Medical Evangelists; died August 5, aged 51, of coronary thrombosis.

Fritz Neuhoff ✦ St. Louis; St. Louis Medical College, 1887; an Associate Fellow of the American Medical Association; associate professor of internal medicine at the St. Louis University School of Medicine; affiliated with St. Mary's Group of Hospitals; died in the Firmin Desloge Hospital in August, aged 85.

Soren Svalheim Norsman, Chicago; College of Physicians and Surgeons, School of Medicine of the University of Illinois, 1900; died August 13, aged 85, of cerebral hemorrhage.

William Asa Onderdonk, Louisville, Ky.; Kentucky School of Medicine, Louisville 1903; member of the American Medical Association; veteran of the Spanish-American War; served on the staffs of St. Joseph Infirmary, SS. Mary and Elizabeth Hospital and the Deaconess Hospital; died August 16, aged 85, of cerebral hemorrhage.

Pedro Nolasco Ortiz, New York; Boston University School of Medicine, 1919; served in the Insular Health Department as chief of the communicable disease bureau and later as health commissioner; visiting professor at the School of Tropical Medicine in San Juan, Puerto Rico, from 1926 to 1930 when he lectured at the Harvard School of Public Health in Boston; affiliated with the Mother Cabrini Memorial Hospital, Park East Hospital and the Mount Sinai Hospital, where he died August 4, aged 60, of peritonitis.

James Bruce Ozier, Amarillo, Texas; Vanderbilt University School of Medicine, Nashville, 1900; member of the American Medical Association; died recently, aged 72, of coronary occlusion.

Frank O. Richards, Santa Ana, Calif.; State University of Iowa College of Homeopathic Medicine, Iowa City, 1901; died recently, aged 79, of arteriosclerotic heart disease.

Robert Elmer Schade, Milwaukee; University of Wisconsin Medical School, Madison, 1928; member of the American Medical Association; served as an examiner for the Selective Service Board during World War II; died recently, aged 45, of fracture of the right clavicle and pulmonary embolism.

Charles Krauth Shanor ✦ Sewickley, Pa.; University of Pittsburgh School of Medicine, 1909; fellow of the American College of Surgeons; affiliated with the Sewickley Valley Hospital; served during World War I; died July 19, aged 66, of carcinoma of the larynx with metastasis to the lungs.

Baxter Hopewell Sigler, Owensboro, Ky.; University of Tennessee College of Medicine, Memphis, 1918; member of the American Medical Association; formerly an officer in the regular U. S. Navy; affiliated with Our Lady of Mercy Hospital and the Owensboro-Daviess County Hospital, where he died July 28, aged 57, of coronary occlusion.

Ralph Douglas Skinner, New York; Baltimore Medical College, 1905; affiliated with the Knickerbocker Hospital, where he died recently, aged 71, of cerebral hemorrhage.

Paul Carrington Spangler ✦ Princeton, W. Va.; College of Physicians and Surgeons, Baltimore, 1912; served on the staff of the Bluefield (W. Va.) Sanitarium; died recently, aged 61, of cardiac aneurysm following coronary occlusion.

Harry Vernon Swick, Erie, Pa.; Temple University School of Medicine, Philadelphia, 1937; member of the American Medical Association; resident in urology at the St. Vincent's Hospital; aged 36; drowned in Lake Erie August 1 as he slipped from his boat while dropping anchor on a fishing trip.

Thomas Francis Tierney, Hudson, Mass.; Baltimore Medical College, 1901; member of the American Medical Association; served during World War I; affiliated with the Clinton (Mass.) Hospital and Marlborough Hospital, Marlboro; died in Wells Beach, Maine, July 24, aged 72, of heart disease.

John Selby Vermillion, Maize, Kan.; College of Physicians and Surgeons, Medical Department Kansas City University, Kansas City, Kan., 1904; for many years city treasurer and treasurer of the school board; member of the American Medical Association; died in St. Francis Hospital, Wichita, August 9, aged 72, of coronary insufficiency and Parkinson's disease

Donald Alfred Waag, Dayton, Ohio; Western Reserve University School of Medicine, Cleveland, 1943; member of the American Medical Association; served during World War II; affiliated with Miami Valley and Good Samaritan hospitals; died July 29, aged 37, of carcinoma.

Wilson Hervey Walters, Fairfield, Maine; Cleveland Homeopathic Medical College, 1898; member of the American Medical Association; affiliated with Sisters' Hospital in Waterville; died July 31, aged 75, of hemiplegia.

Della May Weintraub, New York; New York Homeopathic Medical College and Flower Hospital, New York, 1921; member of the American Medical Association; died July 22, aged 53.

Sidney Colton Williams, Goodman, Miss.; University of Tennessee College of Medicine, Memphis, 1913; served during World War I; formerly affiliated with the Veterans Administration in Jackson and Pittsburgh; died August 1, aged 64, of coronary heart disease.

William Carroll Winstandley ✦ New Albany, Ind.; University of Louisville (Ky.) Medical Department, 1896; past president of the Floyd County Medical Society; served during World War I; on the staff of St. Edward's Hospital, where he died August 28, aged 77, of coronary occlusion.

Louis Wolfson, Philadelphia; Medico-Chirurgical College of Philadelphia, 1899; formerly police surgeon; died in Mount Sinai Hospital August 10, aged 74.

Alexander Francis Wright, Dundee, N. Y.; Trinity Medical College, Toronto, Canada, 1901; member of the American Medical Association; an officer overseas during World War I; served as president of the Dundee Chamber of Commerce and as mayor; on the staff of the Soldiers and Sailors Memorial Hospital in Penn Yan; died in the Arnot Ogden Hospital in Elmira August 21, aged 78, of sarcoma of the mediastinum.

Foreign Letters

BELGIUM

(From Our Regular Correspondent)

Sept. 20, 1949.

Physiology and Pathology of Bones and Joints

The Belgian Foundation for the Study of the Physiology and Pathology of Bones and Joints has been created to stimulate experimental research in physiology and pathology of the osteoarticular system. It will aim to increase the systematic study of the symptoms, diagnosis, clinical aspects and therapy of diseases of the bones, joints, muscles and locomotor apparatus in general; coordinate and disseminate exchanges of information and promote discussions between the laboratory and the clinical staffs, and attempt to find means to aid, support, encourage and reward the research workers. This association hopes to establish connections with all investigators who are interested in the same problems and to make efforts to aid them in proportion to the means at its command.

Legislation to Combat Alcoholism

Belgian legislation concerning the retail sale of alcohol is rather unusual. Nobody is permitted to consume drinks with an alcoholic content in excess of 15 per cent in a public place, such as a café, bar or restaurant. Also, one can neither buy nor carry a quantity of less than 2 liters. Because of the high taxes and other fees involved in this transaction, 2 liters involves considerable expense. This legislation, which was conceived by Minister Van der Velde, had had favorable results in combating alcoholism. Unfortunately, since the war the application of this law has not been carried out with the same strictness as was the case before 1939, and the results of this laxity are becoming evident. The Belgian Society of Neurology and the Society of Mental Hygiene of Belgium have observed an increase in alcoholic psychosis. The Royal Academy of Medicine, at the suggestion of its president, Jules Bordet, has expressed the wish, supported by the Belgium Society of Mental Hygiene, that the legislation of 1919 be strictly enforced.

Instruction in the International Medical Code

The Minister of Public Education, Monsieur Mundeleer, met with the rectors of the four universities of the country and directed that their faculties investigate the possibility of creating a course of instruction on the international medical code in each of the universities. This is in response to an international movement to organize the medical profession to avoid recurrence of the atrocities committed during World War II. Several important medical organizations, such as the World Medical Association and the academies of medicine of Paris and Brussels, have made similar efforts. Dr. Voncken, secretary-general of the International Committee of Military Medicine and Pharmacology, will publish a work concerning this important question, entitled "Attempts at Codification of International Medical Law."

Shortening of the Femur

Continuing the work of the surgical school of Lambotte in Antwerp, which has to his credit technics of great originality and satisfactory results, Dr. Verbrugge presented before the Surgical Society of Belgium a preliminary note on his method of shortening of the femur. To reestablish equality of the two legs the surgeon can either shorten the healthy leg or lengthen the injured leg. The radical intervention consists in shortening the healthy femur. The operation is most satisfactorily done when the patient is between 15 and 20 years old (vigor of bone regeneration, aptitude to muscular readaptation; ease of articular recuperation). Shortening to the extent of 15 to 18 cm. can be carried out.

The operation which Verbrugge presents consists of sectioning the femur at the base of the great trochanter, resection of a part of the lower end, freshening of the circumference of the end of the lower fragment and then insertion of it into the freshened cavity of the superior epiphysis and fixation with metal.

The advantages of his method, he said, are absence of shock and muscular tearing, rapidity of callus formation and the possibility of correction of the axes.

Surgical Treatment of Cutaneous Cancers

In a paper presented to the Belgium Society of Surgery, Cogniaux studied the approach in cases of cutaneous epithelioma and tried to indicate the differential indication for irradiation therapy and for surgery. Irradiation cures the larger number of cutaneous epitheliomas of stage I, for only 8 per cent of these patients succumb as a result of these cancers. However, the results are much less favorable in cases of infiltrating epitheliomas (stage II) or in those which have received irradiation and have relapsed (stage III): in these two categories the mortality rate is about 60 per cent.

The spinocellular, basocellular or mixed histologic types do not seem to be the cause of this, the radiosensitivity of cutaneous epitheliomas being practically in proportion to the employed irradiation.

A clinical study of cutaneous epitheliomas considering the tumor and the area on which it grows will make it possible to determine in advance the lesions which will be refractory to irradiation and which, therefore, must be treated surgically. Cutaneous epitheliomas which are refractory to irradiation can be operated on successfully. Electrosurgery, particularly electrocoagulation, is the method of choice. Exeresis by coagulation resection is employed in so far as is possible; when it appears impossible, coagulation in situ is used to follow the extensions of the tumor.

Since healing by primary intention is generally impossible, it is advisable to close the cutaneous gap by means of free grafts or by plastic methods. Cancers of the vulva require the same therapeutic principles. Electrosurgery is the method of choice; it may be advisable to combine it with radiotherapy, applied to the chain of inguinal lymph nodes.

The nevocarcinomas and the cutaneous sarcomas must be operated on; their resistance to radium treatment depends not on the region in which the tumor is found but on the histologic structure. Extensive electrosurgical treatment eventually combined with radium therapy gives satisfactory results (58 per cent of five year cures), provided the cancer has not yet metastasized. The radiosurgical treatment of cutaneous cancers requires constant cooperation between surgeons and radiologists.

SWITZERLAND

(From Our Regular Correspondent)

GENEVA, Aug. 30, 1949.

Second World Assembly for Mental Health

The Second World Assembly for Mental Health took place in Geneva Aug. 22-27, under the chairmanship of Dr. Rees (London). Dr. Vollenweider (Switzerland) gave the message of welcome from the Swiss government; Dr. Rees discussed the chief aims of the World Federation for Mental Health. The problems of mental prophylaxis need the application of a widespread science through the system of team work. Mental health must be considered as a sort of specialty in the realm of psychiatry, psychology and sociology. It should be possible to synthesize the different professional aims.

The assembly chose as its new chairman Dr. André Repond, director of the Clinic of Malévoz (Switzerland). Dr. Repond

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is a pioneer of mental health in Switzerland, where he founded the first medicopedagogic center, on the model of the American child guidance clinics. In his speech, Dr. Repond emphasized that psychiatrists and psychologists have not yet learned how to produce objective self interest in patients, to permit understanding and control of self in accordance with the principles of modern psychology.

The assembly heard reports on the different aspects of mental hygiene in Switzerland and on the collaboration of the World Federation for Mental Health and the specialized agencies of the United Nations. Dr. Brock Chisholm and Dr. Hargreaves discussed the relations which should exist between the World Federation for Mental Health and the World Health Organization in Geneva in order to allow these two organizations to cooperate in the prevention of crimes, in the study of juvenile delinquency, in the psychologic study of orphans and in the fight against drug addiction. Another problem, an important one and far too often neglected, is the study of mental health in rural districts, in industry and among students.

On the day of relaxation which had been arranged, the members of the conference were driven in coaches to the Clinic of Malévoz which has become the center of most of the social and health activities of the canton of Valais; this canton is formed of many small villages in deep valleys, and the study of the psychologic needs of the population demands a special technic. The conference members were heartily welcomed by the heads of this clinic.

The last day of the program was given over to reports and resolutions presented by study groups which had been organized at the beginning of the assembly and which concerned the following problems: professional teaching of mental health; mental hygiene of the student; education of the public in mental health; mental health in rural districts; mental health aspects of education; religion and mental health; international relations and mental health; psychologic problems of displaced persons, and scientific research in mental health. A well organized teaching system is needed to give training in the basic concepts of mental health. Several members proposed the creation of an international institute of mental health and human relations with a provisional center in Lausanne (Switzerland). The third World Assembly for Mental Health will take place in Paris in 1950.

BRAZIL

(From Our Regular Correspondent)

Rio de Janeiro, Aug. 4, 1949.

Causes of Neonatal Death

Dr. Constantino Mignone of the Department of Pathology and Dr. J. Soares Bicudo of the Department of Obstetrics, School of Medicine, University of São Paulo, have reported a postmortem study of 200 consecutive cases of neonatal death at the Hospital das Clinicas. In eleven instances the cause of death could not be determined. In the remaining 189 cases the causes of death were: prematurity 47, pneumonia and other infections 57, syphilis 18, aspiration and asphyxia 20, obstetric trauma 26, congenital malformations 11, and other causes 10. Under obstetric trauma were listed 17 cases of intracranial hemorrhage, 8 cases of fracture of the parietal bone and 1 case of rupture of the liver. Deaths from congenital malformations included 5 instances of heart defects, 2 of meningoencephalocele, and 1 each of congenital diaphragmatic hernia, anencephaly, omphalocele and esophageal atresia. In "other causes" were included 3 cases of erythroblastosis, 3 cases of congenital debility and 1 case each of acute dyspepsia, ileus paralyticus, hemoperitoneum of undetermined cause and accident during transfusion.

Of the 189 infants the causes of whose death could be ascertained, 72 died on the first day of life, 67 died from the second to the seventh day and 50 from the eighth to the thirtieth day. The causes of death of the 72 who died on the first day were: prematurity 29, pneumonia and other infections 7, syphilis 7, aspiration and asphyxia 15, obstetric trauma 12, malformation 1 and other cause 1.

ARGENTINE

(From Our Regular Correspondent)

Buenos Aires, Oct. 14, 1949.

Action of Sexual Steroids on Experimental Diabetes

Drs. J. T. Lewis, V. G. Foglia and R. R. Rodríguez, at a meeting of the Argentine Society of Biology, reported the results from the daily injection for six months of steroids on diabetes produced experimentally in the white rat by the surgical ablation of 95 per cent of the pancreatic mass. Estrogens (estradiol, estrone, diethylstilbestrol, dienestrol, ethinylestradiol and ethinyltestosterone) exerted an intense preventive action. Cholesterol had a slight preventive effect. Androgens accelerated the appearance of diabetes and made it severer. Ingle observed that daily injection of diethylstilbestrol produced at the beginning glycosuria and hyperglycemia in half the rats deprived of 95 per cent of the pancreas with forced feeding. Dr. R. R. Rodríguez (who read this paper at a meeting of the Argentine Society of Biology, Aug. 4, 1949) found that both hyperglycemia and glycosuria were transitory; after some weeks, glycosuria disappeared and the blood sugar level became normal. These rats were evidently protected against diabetes, since the control animals deprived of 95 per cent of the pancreas which were not given diethylstilbestrol became diabetic in the same period of time.

Estrogens produced pronounced hyperplasia in the islands of Langerhans of the pancreas of the rat, especially the beta cells, with typical granulations, and an increase of the weight of hypophysis and adrenals. There also was observed hyperplasia of the islands of Langerhans and severe lesions in the beta cells of rats that were given injections of androgens. With local implantation of estradiol in the pancreas, the hyperplasia was more intense near the pellet but existed in the whole organ. This was demonstrated in cats, rats and dogs (Drs. A. F. Cardeza and R. R. Rodríguez, Argentine Society of Biology, meeting of October 6).

Argentine Congress of Surgery

At the twentieth Argentine Congress of Surgery in Buenos Aires, October 3-6, the subjects reported and discussed were: bronchiectasia, surgical treatment in adults (Dr. L. Langer, Córdoba, Argentina) and in children (Dr. J. M. Pelliza, Buenos Aires); lumbar sciatica (Dr. G. J. Dickmann, Buenos Aires); thrombophlebitis and phlebothrombosis (W. Chifflet, Montevideo, Uruguay, and J. A. Ferreira, Buenos Aires). Dr. G. Dickmann, neurosurgeon, reported results of the surgical treatment of 120 patients with severe and persistent lumbar sciatica of intervertebral disk origin. Dr. J. Valls (orthopedist) and Dr. J. Diez (general surgeon) discussed the subject.

Personals

Dr. J. C. Ahumada, professor of gynecology, and Dr. T. Padilla, professor of clinical diagnosis, were named members of the National Academy of Medicine of Buenos Aires.

Deaths

Dr. Antonio Cetrángolo, a well known Argentine phthisiologist, died in Buenos Aires, September 21. He was director of Santa María and Ascochinga sanatoriums and of the "Hospital Nacional Central" for tuberculosis patients and president of the Argentine Society of Phthisiology.

Correspondence

NONPARALYTIC POLIOMYELITIS AND MUMPS

To the Editor:—Kilham, Levens and Enders (Nonparalytic Poliomyelitis and Mumps Meningoencephalitis, J. A. M. A. **140**: 934 [July 16] 1949) call attention to the difficulty of making an unequivocal diagnosis of nonparalytic poliomyelitis and state that the diagnosis can be made only on circumstantial evidence. In their paper they reported on 17 patients who were considered to have nonparalytic poliomyelitis during the summer and fall of 1948. Six of these subsequently were proved by serologic tests to have had mumps meningoencephalitis. The correct diagnosis in the 6 patients was made with laboratory procedures worked out by Enders and his associates (Kane, L. W., and Enders, J. F.: Immunity in Mumps: III. The Complement Fixation Test as an Aid in the Diagnosis of Mumps Meningoencephalitis, *J. Exper. Med.* **81**: 137, 1945. Robbins, F. C.; Kilham, L.; Levens, J. H., and Enders, J. F.: The Antihemagglutinin Test in the Diagnosis of Infections by the Mumps Virus, *J. Immunol.* **61**: 235, 1949). Kilham, Levens and Enders' report illustrates the value of the newer serologic and virus studies in patients who have a nonpurulent spinal fluid. However, their paper gives the impression that a clinical differentiation between mumps meningoencephalitis and nonparalytic poliomyelitis is not possible. Unfortunately, the authors do not include the case histories of the patients whose clinical diagnosis of poliomyelitis was incorrect.

There are many differences between mumps meningoencephalitis and nonparalytic poliomyelitis: The seasonal incidence of mumps in the United States, as tabulated by the United States Public Health Service, is greatest in the spring of the year, and although sporadic cases do occur in the late summer these are unusual. Mumps is prevalent annually in most localities. Poliomyelitis, on the other hand, makes its appearance somewhat later in the year, the exact time varying in different localities. The seasonal incidence is the same in a given locality whenever the disease appears, and a heavy outbreak in two successive years in a given area is unusual. There is little poliomyelitis after an epidemic subsides. Thus the two diseases actually do not occur simultaneously, though conceivably the end of a mumps outbreak may coincide with the onset of an epidemic of poliomyelitis.

The statement is made by Kilham, Levens and Enders, "No essential difference was observed between the clinical signs and symptoms presented by the two groups of patients." This is at variance with the usual findings in mumps meningoencephalitis and nonparalytic poliomyelitis. A careful history of exposure to mumps should be sought, and a daily inspection of the patient, including palpation of the salivary glands and testes, is important. Cases of mild disease do occur, but usually the patient with mumps meningoencephalitis is drowsy, if not unconscious, and resembles a patient with one of the forms of bacterial meningitis. He is often treated with sulfonamide preparations or antibiotics because the condition which he presents is so severe that it is imprudent to withhold therapy pending an accurate laboratory diagnosis. The patient with nonparalytic poliomyelitis, on the other hand, may be somewhat drowsy, but can be roused and presents a number of positive symptoms and signs which are not found in patients with mumps. These include inability to flex the spine, called originally by Aycock, I believe, "the audible sign." The general body pains, hyperesthesias and anxiety common in poliomyelitis are absent in mumps. There are also numerous other minor differences between poliomyelitis and mumps involvement of the central nervous system, such as the frequent high spinal fluid leukocyte count in mumps.

It must be remembered that the only way in which a positive clinical diagnosis of poliomyelitis can be made is with the appearance of definite muscle weakness or bulbar involvement. These do not occur (or rarely) in mumps. Even the isolation of poliomyelitis virus from the stool does not necessarily prove that the patient has or has had the clinical disease, for it is conceivable that patients with mumps may be carriers of poliomyelitis virus just like anyone else during an epidemic. The authors infer that, because a low recorded incidence of the paralytic form of poliomyelitis occurred during 1948 in Massachusetts, one might therefore explain the unusually high incidence of mumps meningoencephalitis in their patients with supposed nonparalytic poliomyelitis. If this inference were correct, one would also expect large numbers of diagnostic errors in years when paralytic poliomyelitis is prevalent. This is not the case. The authors' point is well taken, however, that in nonepidemic years one should hesitate to make a diagnosis of nonparalytic poliomyelitis without due consideration of other possibilities, such as mumps meningoencephalitis. One cannot agree with the statement, "Without a definite history of this sort [that is, contact with a patient with poliomyelitis] the mere occurrence of aseptic lymphocytic meningoencephalitis during the late summer and early fall, when poliomyelitis may be epidemic, is insufficient to warrant even a tentative diagnosis of the nonparalytic form of the disease." It is more prudent for the physician to consider the most likely diagnosis—poliomyelitis—even without a history of contact, for even in epidemic years, history of direct exposure is obtained in but a small percentage of patients with poliomyelitis. The differences between the two diseases are being pointed out in order that proper therapeutic measures, such as suction apparatus and a respirator, may be at hand when a patient has nonparalytic poliomyelitis, for poliomyelitis is a treacherous disease which may progress rapidly and requires prompt treatment to prevent fatal termination.

ALFRED E. FISCHER, New York.

OXYGEN REQUIREMENTS FOR PATIENTS TRAVELING BY AIR

To the Editor:—Physicians often are asked for instructions and advice to patients who are planning air travel. Most medical men are familiar with the facilities provided in modern transport aircraft, but startling instances of misunderstanding by patients or physicians sometimes occur. Recently, for example, an elderly woman boarded a transcontinental plane with a nurse and her oxygen tank. One of her astonished fellow travelers reported, "The nurse continually manipulated the tank, which was 3½ feet high and at least a foot in diameter, with plyers and screw drivers."

The cabins of modern transport planes which travel at altitudes above 10,000 feet are pressurized to simulate atmospheric conditions at 8,000 to 10,000 feet; over many routes the cabin pressure is maintained equivalent to lower altitudes of 5,000 to 6,000 feet. Most air lines will provide supplementary oxygen for emergency use should the passenger request it; this is supplied on a fixed diaphragm basis and at a liter per minute rate which compensates for the deficit of atmospheric oxygen cabin altitude or, in nonpressurized planes, at the altitude at which the plane is flying. Maximum delivery is generally 2.25 liters per minute, which would be enough to provide for most persons an oxygen concentration considerably in excess of that at sea level, even when the cabin pressure is at the 10,000 foot level. Thus, if a passenger can take up the oxygen provided, the airlines can maintain conditions which are not more severe than ground conditions.

However, if the air traveler requires oxygen in therapeutic doses or other special services, arrangements should be made with the air line before the flight, since such facilities are not routinely available. The physician should call the passenger agent to request these special services. If my experience, and

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that of my professional associates, constitutes a fair sample, the physician requesting special care will be gratified by the effective and wholehearted cooperation he will receive in insuring a safe and comfortable trip for his ambulatory patient.

W. R. Stovall, M.D.
Chief, Medical Division, Civil Aeronautics Administration, Washington, D. C.

Council on Medical Education and Hospitals

HOSPITAL FACILITIES FOR ALCOHOLIC PATIENTS

Report of Survey by Council on Medical Education and Hospitals

It has been estimated that 50,000,000 persons in the United States use alcoholic beverages and that of this number 6 per cent, or 3,000,000, become excessive drinkers; in the latter group nearly one fourth, or 750,000, have been said to be chronically alcoholic.[1] These estimates, it will be agreed, represent a major problem in relation to both social welfare and public health. It is recognized that the excessive drinker creates both a social and a legal problem; yet it is the growing view that the alcoholic person with his associated disorders should be regarded primarily as a sick person. While the medical approach emphasizes health needs, due consideration is also given to the educational, religious, social, economic, legal, psychologic and other factors involved in the problem of alcoholism.

With reference to the medical aspects of alcoholism, the following resolution was adopted at the Conference on Medical, Legal and Social Approaches to the Problem of Inebriety held in New York City, Jan. 8, 1947:

> *Be It Resolved Therefore* That the American Medical Association, the American College of Surgeons and other professional standardizing bodies be requested to review and/or establish minimum standards of hospitalization for alcoholic patients with the view toward: (1) establishing criteria for the effective care and treatment of alcoholic patients, and (2) encouraging hospitals to adopt a more favorable attitude towards the hospitalization of such patients; and that the resolution be transmitted to the officials concerned for their appropriate action.

This resolution sponsored jointly by the New York Academy of Medicine and the Research Council on Problems of Alcohol was given careful consideration by the Council on Medical Education and Hospitals at its meeting in Atlantic City June 1947, at which time the following action was reported to the Board of Trustees of the American Medical Association:

> The Council on Medical Education and Hospitals recognizes the importance of improving the standards and facilities for the hospitalization and care of alcoholic patients. As an initial step in the study of this problem, the Council in its next Annual Census of Hospitals, will obtain further information regarding the extent to which facilities for the treatment and care of alcoholic persons have been provided in general, psychiatric and other hospitals throughout the country. This information may then serve as a basis for further studies or action in cooperation with other agencies that are likewise concerned in the advancement of this program.

In conformity with this action the Council, at the end of 1947, requested the following information in the Annual Survey of Hospitals registered by the American Medical Association:

1. Do you admit patients for the treatment of acute alcoholism? for chronic alcoholism?
2. If so, how many beds are available for this purpose?
3. How many patients were admitted for the treatment of alcoholism last year?

Of a total of 6,276 hospitals included in this survey, 1,718 reported that alcoholic patients are accepted for treatment. These include 1,354 general hospitals, 291 nervous and mental institutions and 73 appearing under other classifications, as shown in table 1.

1. The Scientific Approach to the Problem of Chronic Alcoholism, by the Research Council on Problems of Alcohol, New York City.

On the basis of control, 202 are listed as federal hospitals, 396 under state, city or county control, 787 as church or other nonprofit associations and 330 as proprietary hospitals. A further subdivision of the hospitals classified by control is shown in table 2.

With reference to the question "Do you admit patients for the treatment of acute alcoholism? For chronic alcoholism?" 783 hospitals reported the admission of acutely alcoholic persons only and 123 accept only chronically alcoholic persons, while 812 admit patients of both classifications. Combining these figures, one notes that acutely alcoholic persons are admitted in 1,595 hospitals and chronically alcoholic persons in 935.

Although 1,718 hospitals reported the acceptance of alcoholic patients, only 448 indicated the number of beds available for this purpose. The report of 4,026 beds, therefore, should not be interpreted as representing the total facilities currently available in the hospitals in this country. At least one or more beds would also be available in each of the 1,270 hospitals which, according to the report, admit alcoholic patients but did not specify the number of beds. It may be of interest to note that, of the beds specifically allotted for alcoholic patients, 1,537 are in general hospitals, 2,117 in nervous and mental institutions and 372 in other hospitals. In relation to ownership or control, it is found that the federal hospitals report 660 beds, those under state, city and county control 1,616, the church and other nonprofit associations 730 and the proprietary hospitals 1,020. Further information on the distribution of beds is contained in tables 1, 2 and 3.

The report on the number of admissions was more complete, however, as 1,287 hospitals furnished information of this type. From this group came reports of 77,841 admissions for the treatment of acute and chronic alcoholism in the preceding year. These included 43,812 in general hospitals, 25,842 in nervous and mental institutions and 8,187 in other hospitals. The federal hospitals admitted 10,460, the state, city and county

Table 1.—*Hospitals Admitting Alcoholic Patients (Classified by Type of Service)*

Type of Service	Number of Hospitals	Beds for Alcoholic Patients	Alcoholic Patients Admitted
General	1,354	1,537	43,812
Nervous and mental	291	2,117	25,842
Tuberculosis	5	5	18
Industrial	3	...	21
Alcoholic	4	83	2,350
Convalescent and rest	3	20	439
Chronic	3	8	74
Drug	4	113	3,011
Cancer	1	1	3
Institutions	50	142	2,181
Totals	1,718	4,026	77,841

Table 2.—*Hospitals Admitting Alcoholic Patients (Classified by Control)*

Control	Number of Hospitals	Beds for Alcoholic Patients	Alcoholic Patients Admitted
U. S. Army	70	147	2,017
U. S. Navy	31	102	408
U. S. Public Health Service	26	56	1,383
Veterans Administration	56	282	6,412
Indian Affairs	21	4	90
Other federal	4	9	170
State	149	1,157	7,988
County	124	225	6,080
City	108	232	16,653
City-County	15	2	702
Nonprofit associations	426	442	10,571
Church	361	288	7,418
Individual	164	424	6,559
Partnership	56	139	2,337
Corporation	110	457	9,074
Totals	1,718	4,026	77,841

hospitals 31,423, the church and other nonprofit associations 17,989 and the proprietary group 17,969. Again reference should be made to tables 1, 2 and 3, which show a further distribution of admissions by states, type of hospital service and control.

Lists of hospitals offering facilities for the treatment of alcoholism have been prepared showing the name of each hospital, location, type of service, control, the number of beds

TABLE 3.—*Hospitals Admitting Alcoholic Patients (Classified by States)*

State	No. of Hospitals	Admit Acutely Alcoholic Patients	Admit Chronically Alcoholic Patients	Beds for Alcoholic Patients	Alcoholic Patients Admitted
Alabama	34	33	15	25	1,453
Arizona	20	20	8	19	239
Arkansas	16	14	7	24	697
California	99	88	67	6.8	6,101
Colorado	29	23	19	37	1,200
Connecticut	22	20	17	159	1,013
Delaware	5	4	3	53	5
District of Columbia	6	6	5	58	1,922
Florida	39	37	23	112	2,154
Georgia	36	34	18	109	2,354
Idaho	17	17	5	3	160
Illinois	85	79	53	174	4,938
Indiana	29	25	14	43	441
Iowa	39	35	15	31	1,700
Kansas	43	38	25	50	542
Kentucky	22	22	9	100	1,021
Louisiana	16	16	8	21	624
Maine	12	10	9	56	172
Maryland	27	27	17	91	1,493
Massachusetts	37	33	24	141	2,766
Michigan	72	67	43	76	4,225
Minnesota	61	58	26	168	1,276
Mississippi	28	28	12	52	1,091
Missouri	39	39	19	103	1,755
Montana	29	27	15	23	508
Nebraska	34	31	17	126	698
Nevada	10	9	4	10	62
New Hampshire	10	10	3	..	114
New Jersey	36	33	19	63	2,758
New Mexico	21	21	11	9	248
New York	121	115	67	262	12,175
North Carolina	37	34	21	126	1,819
North Dakota	24	23	15	40	336
Ohio	52	49	28	153	1,929
Oklahoma	25	24	10	27	445
Oregon	22	20	12	25	1,515
Pennsylvania	77	64	43	93	3,658
Rhode Island	5	5	4	..	126
South Carolina	17	16	8	..	549
South Dakota	21	18	14	12	202
Tennessee	30	29	12	53	1,804
Texas	107	104	42	125	2,516
Utah	8	8	4	28	116
Vermont	14	13	6	9	211
Virginia	34	34	24	133	2,700
Washington	40	36	19	166	2,074
West Virginia	23	22	13	44	449
Wisconsin	73	65	42	74	1,979
Wyoming	15	14	9	42	427
Totals	1,718	1,595	935	4,006	77,841

available for alcoholic patients, number of admissions and whether the hospital accepts acutely alcoholic persons, chronically alcoholic persons, or both. As indicated in the Council's report to the Board of Trustees, the present survey of hospital facilities should be regarded as an initial step in the study of the problem of alcoholism. This should be supplemented, however, by further studies and planning in accordance with the initial resolution, which aims at the establishment of specific criteria for the more effective care and treatment of alcoholic patients. To reach this purpose will require the combined efforts of all who are interested in seeking a reasonable solution of the problem of alcoholism.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS. *Parts I and II*, Feb. 13-15. Centers where there are approved medical schools and five or more candidates. Exec. Sec., Mr. E. S. Elwood, 225 S. 15th Street, Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF NEUROLOGICAL SURGERY: *Oral.* Chicago, June 3. Final date for filing applications is Jan. 1. Sec., Dr. W. J. German, 789 Howard Ave., New Haven.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC. *Written and Review of Case Histories. Part I.* Various Centers. Feb. 3. Final date for filing applications is Nov. 5. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh.

AMERICAN BOARD OF OPHTHALMOLOGY: Boston, April. Sec. Dr. Edwin B. Dunphy, 56 Ivie Rd., Cape Cottage, Maine.

AMERICAN BOARD OF ORTHOPAEDIC SURGERY. *Part II*, New York City, Feb. 9-10. Sec. Treas., Dr. Harold A. Sofield, Room 1856, 122 S. Michigan Ave., Chicago.

AMERICAN BOARD OF PLASTIC SURGERY: Examinations are given in June and November of each year in the home town of applicants. Sec.-Treas., Dr. Louis T. Byars, 400 Metropolitan Bldg., St. Louis, Mo.

AMERICAN BOARD OF PROCTOLOGY: *Examination in Anorectal Surgery.* Philadelphia, Nov. 12. Sec.-Gen., Dr. Louis A. Buie, 102-110 Second Ave., S.W., Rochester, Minn.

AMERICAN BOARD OF UROLOGY: *Written.* Various Centers, Dec. 3. *Oral and Clinical.* Chicago, Feb. 11-15. Sec., Dr. Harry Culver, 7935 Sunnyside Road, Minneapolis 21.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 27-29. Sec., Dr. D. G. Gill, 519 Dexter Avenue, Montgomery.

ARKANSAS: * *Examination.* Little Rock, Nov. 3-4. Sec., Dr. Joe Versee, Harrisburg. *Eclectic.* Little Rock, Nov. 3. Sec., Dr. Clarence H. Young, 1415 Main St., Little Rock.

COLORADO: * Denver, Jan. 3-6. Sec., Dr. George H. Gillen, 831 Republic Building, Denver.

CONNECTICUT: * *Examination.* Hartford, Nov. 8-9. Secretary to the Board, Dr. Creighton Barker, 160 St. Ronan Street, New Haven. *Homeopathic.* Derby, Nov. 8-9. Sec., Dr. Donald A. Davis, 38 Elizabeth St., Derby.

DELAWARE: *Examination.* Dover, Jan. 10-12. *Reciprocity.* Dover, Jan. 19. Sec., Dr. J. S. McDaniel, 229 State Street, Dover.

DISTRICT OF COLUMBIA: * *Examination.* Washington, Nov. 14-15 Sec., Mr. Paul C. Foley, Admin. Asst., 4130 E. Municipal Bldg., Washington.

FLORIDA: * *Examination.* Jacksonville, Nov. 27-29. Sec., Dr. Frank D. Gray, 12 N. Rosalind Ave., Orlando.

GUAM: *Endorsement.* Agana, Last Friday of each month. Sec., Capt. C. K. Youngkin, Dept. of Public Health, Guam, % F.P.O., San Francisco.

HAWAII: *Examination.* Honolulu, Jan. 9-12. Sec., Dr. I. L. Tilden, 881 S. Hotel St., Honolulu.

IDAHO: Boise, Jan. 9. Exec. Sec., Mr. Armand L. Bird, 305 Sun Building, Boise.

INDIANA: *Examination.* Indianapolis, June. Sec., Dr. Paul R. Tindall, 1138 K. of P. Bldg., Indianapolis.

IOWA: * *Examination.* Des Moines, Dec. 5-7. *Reciprocity.* Des Moines, Nov. 7, Dec. 5. Sec., Dr. M. A. Royal, 506 Fleming Bldg., Des Moines.

KANSAS: Topeka, Dec. 8-9. Sec., Dr. J. F. Hassig, 905 N. Seventh St., Kansas City.

KENTUCKY: Louisville, Dec. 12-14. Sec., Dr. Bruce Underwood, 620 South Third Street, Louisville 2.

LOUISIANA: New Orleans, Dec. 8-10. Sec., Dr. Roy B. Harrison, 1507 Hibernia Bank Building, New Orleans 12.

MAINE: Portland, Nov. 8-9. Sec., Dr. Adam P. Leighton, 192 State Street, Portland.

MARYLAND: *Examination.* Baltimore, Dec. 13-16. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore 1. *Homeopathic. Examination.* Baltimore, Dec. 13-14. Sec., Dr. John A. Evans, 612 West 40th St., Baltimore.

MASSACHUSETTS: Boston, Nov. 15-18. Sec., Dr. George L. Schadt, 413 East State House, Boston.

MISSISSIPPI: *Reciprocity.* Jackson, December. Sec., Dr. Felix J. Underwood, State Board of Health, Jackson 113.

NEVADA: Carson City, Nov. 7. Sec., Dr. George H. Ross, 112 N. Curry St., Carson City.

NEW HAMPSHIRE: Concord, March 8-9. Sec., Dr. John S. Wheeler, 107 State House, Concord.

NORTH DAKOTA: *Examination.* Grand Forks, Jan. 4-6. *Reciprocity.* Jan. 7. Sec., Dr. C. J. Glaspel, Grafton.

OHIO: *Examination.* Columbus, Dec. 12-14. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: * *Examination.* Oklahoma City, June 7-8. Sec., Dr. Clinton Gallaher, 813 Braniff Building, Oklahoma City.

UNIVERSITY OF MICHIGAN LIBRARIES

OREGON: * *Endorsement.* Portland, Nov. 4. *Written.* Portland, January. Exec. Sec., Mr. Howard I. Bobbitt, 608 Failing Building, Portland 4.

PENNSYLVANIA: *Examination.* Philadelphia or Harrisburg, January. Acting Sec., Mrs. Marguerite G. Steiner, 351 Education Building, Harrisburg.

PUERTO RICO: *Examination.* Santurce, March 7. Sec., Mr. Luis Cueto Coll, Box 3717, Santurce.

SOUTH CAROLINA: *Examination.* Columbia, Nov. 8-9. *Reciprocity.* First Monday of every month. Sec., Dr. N. B. Heyward, 1329 Blanding St., Columbia.

SOUTH DAKOTA: * Sioux Falls, Jan. 17. Sec., Dr. C. E. Sherwood, 300 First National Bank Building, Sioux Falls.

UTAH. *Examination.* Salt Lake City, June. Dir., Dr. Frank E. Lees, 324 State Capitol Building, Salt Lake City.

VIRGINIA: *Examination.* Richmond, Dec. 2-3. *Reciprocity.* Richmond, Dec. 1. Sec., Dr. K. D. Graves, 631 First St., S.W., Roanoke.

WASHINGTON: * Seattle, January. Director, Department of Licenses, Mr. Edward C. Dohm, Olympia.

WISCONSIN: * *Examination.* Madison, Jan. 10-12. Sec., Dr. C. A. Dawson, River Falls.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ARIZONA: *Examination.* Tucson, Dec. 20. Sec., Mr. Francis A. Roy, Science Hall, University of Arizona, Tucson.

COLORADO: *Examination.* Denver, Dec. 7-8. Sec., Esther B. Starks, 1459 Ogden Street, Denver.

FLORIDA: *Examination.* Gainesville, Nov. 5. Sec., Mr. M. W. Emmel, University of Florida, Gainesville.

NEW MEXICO: *Examination.* Santa Fe, Nov. 6. Sec., Miss Marguerite Kilkenny, 110 W. Houghton St., Santa Fe.

OKLAHOMA: *Examination.* Oklahoma City, April 11. Sec., Dr. Clinton Gallaher, 813 Braniff Building, Oklahoma City.

OREGON: Portland, Dec. 3. Sec., Mr. Charles D. Byrne, State Board of Higher Education, Eugene.

SOUTH DAKOTA: Vermillion, Dec. 2-3. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

TENNESSEE: *Examination.* Memphis, Dec. 30-31. Sec., Dr. O. W. Hyman, 874 Union Avenue, Memphis 3.

WASHINGTON: Seattle, January. Sec., Department of Licenses, Mr. Edward C. Dohm, Olympia.

WISCONSIN: Milwaukee, Dec. 3. Madison, April 1. Sec., Prof. W. H. Barber, Ripon College, Ripon.

Medical Motion Pictures

FILM REVIEW

The Circulation in the Fetus and in the Newborn. 16 mm., color, sound, 350 feet, showing time nine minutes. This is part of a series on Congenital Heart Disease prepared under the direction of George H. Humphreys II, M.D., Director of Surgical Service, Presbyterian Hospital, New York and Professor of Surgery, College of Physicians and Surgeons. Produced in 1949 by and procurable on rental or purchase from Sturgis-Grant Productions, Inc., 314 East Forty-Sixth Street, New York 17.

This is an animated film showing diagrammatically the differences in circulation during the fetal life as contrasted with life after birth. It shows the role of the placenta in the fetal circulation and the dependence of the fetus on the maternal body. The changes in the circulatory system at term, with the lungs of the baby starting to function, are graphically illustrated. The various degrees of oxygenation are shown throughout the film by differences in the red and blue color of the different vessels.

This film is recommended as a teaching film for general practitioners who may have forgotten some of the intricacies of the changes in circulation and in birth, for residents and for medical students during their course in obstetrics.

The animation and narration are well done.

Alcohol and the Human Body. 16 mm., black and white, sound, 480 feet (1 reel), showing time ten minutes. Prepared in collaboration with Anton J. Carlson, M.D., Ph.D., Scientific Director, The Research Council on Problems of Alcohol, Chicago. Produced in 1949 by and procurable on rental or purchase from Encyclopaedia Britannica Films, Inc., 1150 Wilmette Avenue, Wilmette, Ill.

This film undertakes to make clear, without resorting to abstruse terminology, the course that ethyl alcohol takes when it enters the human body in the form of beverages. The preparation of beer, wine and whiskey is described; their relative strengths are indicated, and their effects on behavior are suggested. Animated drawings show the absorption of the alcohol from the stomach and small intestine, the partial change to acetic acid in the liver, the circulation of the unchanged fraction in the blood and the gradual depression of successive parts of the brain. The nutritional consequences of chronic alcoholism are set forth simply and without exaggeration, and the proper hospital care of a patient in acute intoxication is illustrated.

This film is a cautious and objective introduction to a difficult subject. The narration appears to have been written with extraordinary care and every statement is undeniably unemotional.

This production will be found to be instructive by any group over the age of 12, but would probably be most useful at high school and young adult ages. The chemistry and physiology are elementary and could be understood by those without special training in these fields.

The photography, animation and narration are excellent.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Workmen's Compensation Acts: Compensability of Death from Tularemia.—The petitioner filed a claim for a compensation award after the death of her husband. From the granting of an award by the commissioner, which was sustained in the trial court, the defendant appealed to the Supreme Court of Iowa.

The petitioner's husband was employed by the defendant to skin and stretch hides from fur-bearing animals. He worked with a slight cut or scratch on his finger. This cut developed into an "indolent ulcer," described by his physician as a manifestation of tularemia; all the other symptoms of tularemia developed, and he subsequently died of the disease. The trial court, in affirming an award for the petitioner, held that it was immaterial whether or not the cut on the deceased's finger had been received in the course of his employment and said ". . . the employment is shown without dispute to be one in which one might be exposed to tularemia. . . . It was the opinion of the doctors who testified that the deceased contracted the disease while engaged in the company's work."

The great majority of workmen's compensation acts, said the Supreme Court of Iowa, are limited to payment of compensation benefits for accidental injury. Our statute does not contain the "accidental" limitation. The benefits accrue from a personal injury arising out of and in the course of the workman's employment. The terms "injury" and "personal injury" are defined in the Iowa act as not to "include a disease unless it shall result from the injury." We have held, the court continued, that the injury may be a disease which is not occupational, that it may be an aggravation of injuries which in their origin were apart from the employment and that, in order to prove that the employee received a personal injury in his work, it is not necessary that there be proof of some special incident or unusual occurrence. Since it is not contended that the tularemia was an occupational disease, the only inquiry is whether the injury (in this case the disease of tularemia of which he died) arose out of and in the course of his employment. The doctors were emphatic in their opinions that the deceased contracted tularemia by the entry of the germ into the cut on his finger while he worked for the defendant, skinning animals which were infected with such disease. The medical testimony as to the probable origin of the disease was supplied with abundance. According to the doctors, the cut was the channel of entry of the tularemia germ. We are convinced, the Supreme Court concluded, that the record amply sustains the commissioner's finding that the disease arose out of and in the course of employment. Accordingly the commissioner's award in favor of the petitioner was affirmed.—*Ford et al. v. Goode et al., 38 N.W. (2d) 158 (Iowa, 1949).*

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1938 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Journal of Clinical Pathology, Baltimore

19:401-500 (May) 1949

Universal Serologic Reaction with Lipid Antigen: III. In Syphilis. R. L. Kahn and E. B. McDermott.—p. 401.
Id.: IV. In Lepromatous Leprosy. R. L. Kahn, B. J. Baribeau and F. T. Villalon.—p. 408.
Id.: V. In Malaria. R. L. Kahn.—p. 414.
Id.: VI. In Tuberculosis. R. L. Kahn.—p. 419.
Diagnostic Significance and Laboratory Methods in Determination of the 17-Ketosteroids. R. L. Landau.—p. 424.
Anti-Rh Agglutinins: Their Clinical Significance. A. W. Frisch and V. Jackets.—p. 435.
Mucopolysaccharide Content of Skin in Localized (Pretibial) Myxedema. H. E. M. Watson and R. H. Pearce.—p. 442.
Determination of Anti-Tryptic Activity in Human Plasma. B. B. Wells, H. N. Marvin and M. Waldvogel.—p. 448.

American Journal of Diseases of Children, Chicago

77:561-702 (May) 1949

Relation of Hemolytic Streptococcus to Rheumatic Fever: IV. Effect of Streptococcic Spreading Factor in Rheumatic Patients and Others. T. N. Harris and S. Friedman.—p. 561.
Vitamin A Absorption in the Newborn. A. E. Sobel, L. Besman and B. Kramer.—p. 576.
Irregularities of Breathing in the Newborn Period. P. J. Howard and A. R. Bauer.—p. 592.
Standard, Unipolar Limb and Precordial Leads in Children and Adolescents with Inactive Rheumatic Fever. M. F. Dunning and A. G. Kuttner.—p. 610.
Respiratory Air Flow Patterns in Children. R. Kaye, J. L. Whittenberger and L. Silverman.—p. 625.
Pediatric Report on Medical Conditions in the Virgin Islands. H. N. Sanford.—p. 662.
Pediatric Report on Medical Conditions in Puerto Rico. H. N. Sanford.—p. 665.

Archives of Internal Medicine, Chicago

83:477-592 (May) 1949

*Iron Overload (Hemosiderosis) Aggravated by Blood Transfusions. E. E. Muirhead, G. Crass, F. Jones and J. M. Hill.—p. 477.
Concurrent Herpes Zoster and Chickenpox: Report of Case. F. Fetter and T. G. Schnabel.—p. 502.
Disseminated Coccidioidomycosis and Lymphohematogenous Tuberculosis: Report of Case. B. Hyde and L. Hyde.—p. 505.
Present Status of Insulin Resistance: Report of Case with Autopsy. E. A. Haunz.—p. 515.
*Acute Renal Insufficiency: Comparison of Use of Artificial Kidney, Peritoneal Lavage and More Conservative Measures in Its Management. E. E. Muirhead, J. Vanatta and A. Grollman.—p. 528.
Colloid Osmotic Pressure in Chronic Hepatitis. M. Bjørneboe, C. Brun and F. Raaschou.—p. 539.
Activity of Carbon Anhydrase in Blood: Study of Patients with Dyspnea Consequent to Chronic Cardiac and Pulmonary Disease. M. D. Altschule and H. D. Lewis.—p. 547.
Gastroenterology: Review of Literature from July 1947 to July 1948. J. B. Kirsner, W. L. Palmer, W. E. Ricketts and others.—p. 552.

Iron Overload Aggravated by Blood Transfusions.—Muirhead and associates point out that, with the advent of blood banks and the greater confidence in blood transfusion therapy, larger volumes of blood are being administered. Under special conditions it is possible for prolonged transfusion therapy to be associated with a prominent iron overload of various tissues. In certain organs this metallic excess is associated with parenchymatous damage. The present discussion treats especially of the iron overload of anemic patients requiring transfusions over prolonged intervals. Five closely studied cases in which autopsies were subsequently performed are reported. The 5 cases represent three different kinds of severe anemia. There are 2 instances of acquired hemolytic anemia that failed to respond to splenectomy. In 2 cases the picture of "aplastic anemia" was present. The fifth case was one of classic pernicious anemia with repeated relapses due to failure of the patient to maintain liver therapy. In each of these 5 patients excessive iron deposits were evident throughout the body. It is apparent that blood transfusions by by-passing the mucosal absorption mechanism afford a rich source of excess iron (0.25 Gm. per 500 cc.). In cases 1, 2 and 5 the amount of infused iron seemed to account only for the liver iron content. The excessive iron deposited in other tissues requires an explanation. Since the patients received oral iron therapy during part of their illness and since the dietary intake throughout most of the illnesses was adequate, it appears that absorption of iron via the mucosa occurred despite an already existing excess of iron. The authors conclude that the outlook for patients that are sustained hematologically by prolonged blood transfusion therapy does not appear good. Two complications seem likely to develop: (*a*) frequent reactions to the infused blood, with a more rapid dissipation of the infused erythrocytes, and (*b*) iron overload of body tissue, mainly the liver.

Management of Acute Renal Insufficiency.—According to Muirhead and collaborators reports in the current scientific literature as well as in the lay press describing the use of the artificial kidney or peritoneal lavage in the treatment of acute oliguria or anuria give an erroneous impression of their wide field of usefulness as well as of the success of their application. The authors' investigations demonstrated many inherent dangers which previous investigators had failed to note. They do not aim to discredit the use of an artificial kidney, peritoneal lavage or other measures for removing waste products for certain patients. They wish to emphasize that the procedure, as at present available, has not been proved safe and that many patients so treated can be more effectively and more safely handled by simpler measures. They are at present engaged in attempting to perfect a safe artificial kidney. Until a given procedure has been proved capable of consistently prolonging the life of animals that have undergone nephrectomy, it is hazardous to apply it to a patient. It is preferable to rely on the normal course of events which results, in most cases, in spontaneous recovery of renal function, if the patients are not overloaded with salt and water in the vain attempt to stimulate urine production. The application of the artificial kidney of Kolff in dogs that have been subjected to nephrectomy failed to prolong life, despite an adequate removal of urea from the animals. Clinical data are presented which demonstrate that, contrary to current views, recovery may occur even after periods of anuria as long as eight days. A review of the literature indicates that patients surviving the application of the artificial kidney or peritoneal lavage would have probably survived without their application. The use of safer procedures for the treatment of acute renal failure is advocated.

Archives of Pathology, Chicago

47:411-516 (May) 1949

Effects of Various Degrees of Protein Depletion on Histologic and Clinical Structure of Rat Liver. E. R. Jaffé, Eleanor M. Humphreys, E. P. Benditt and R. W. Wissler.—p. 411.
Proliferation of Epithelium of Intrahepatic Bile Ducts in Parabiotic Rats Following Obstruction of Bile Ducts. I. T. Zeckwer.—p. 424.
Right Aortic Arch with Vascular Ring Constricting Esophagus and Trachea: Report of 2 Cases. B. Halpert, W. T. Snoddy, K. E. Bohan and C. L. Freede.—p. 429.
Acute Lesions in Weaning Rats Fed 4-Aminopteroylglutamic Acid. H. B. Ritter and J. J. Oleson.—p. 435.
Effects of Administration of Vitamin E Concentrate and Cholesterol and Bile Salt on Aorta of Rat. W. Marx, Love Marx, Emily R. Meserve and others.—p. 440.
Thrombotic Thrombopenic Purpura Caused by Iodine: Report of Case. W. E. Ehrich and J. Seifter.—p. 446.
Relations Between Volumes of Closed Hypothermal Cerebral Lesions and Symptoms in Rabbits. C. B. Taylor, G. M. Hass and J. E. Maloney.—p. 450.
Effect of Heat on Nutritive Value of Lactalbumin: Chemical and Morphologic Changes. P. Rizzo, Ruth M. Davis and A. H. Smith.—p. 464.
Cardiac Hypertrophy: Immediate Response to Starling's Law of Increased Energy Output of Heart. Cecil A. Krakower and Helen E. Heino.—p. 475.
Cartilage in Kidney. J. A. Bigler and W. P. Killingsworth.—p. 487.
Late Effects of Radium and Plutonium on Bone. Margaret A. Bloom and W. Bloom.—p. 494.

UNIVERSITY OF MICHIGAN LIBRARIES

Delaware State Medical Journal, Wilmington

21:67-86 (May) 1949

Diabetes Mellitus and Arteriosclerosis. E. M. Bohan.—p. 67.
Gastrojejunocolic Fistula: Case Report. P. Alava and W. W. Lattomus. —p. 70.
Radical Surgery for Advanced Cancer. G. S. Serino.—p. 74.
Local Health Boards in Delaware. G. J. Boines.—p. 78.
Time to Stand Up and Be Counted. G. F. Lull.—p. 82.

Florida Medical Association Journal, Jacksonville

35:673-740 (May) 1949

Nasal Sinusitis: Clarification of Certain Problems of General Interest. A. R. Hollender.—p. 691.
Ureteral Calculi in Children. A. J. Butt.—p. 696.
Venereal Diseases: Diagnosis and Treatment. R. F. Sondag.—p. 699.
Role of Quinine and Urea Hydrochloride in Proctology. R. V. Terrell and C. C. Chewning Jr.—p. 704.

Iowa State Medical Society Journal, Des Moines

39:187-230 (May) 1949

Diagnosis and Surgical Treatment of Pulmonary Stenosis. W. J. Potts. —p. 187.
Ritter's Disease. J. N. Lande.—p. 189.
Pulsion Diverticulum of Esophagus. L. T. Palumvo.—p. 191.
Accessory Scrotum, Posteriorly Located: Review of Literature and Report of Case. W. W. Daut and R. V. Daut.—p. 194.
Veterans Administration Problem: Survey of 200 Consecutive Cases. T. B. Throckmorton.—p. 196.

39:231-278 (June) 1949

President's Address. J. E. Reeder.—p. 231.
President-Elect's Address. N. G. Alcock.—p. 231.
Saddle Block Anesthesia in Pelvic Deliveries: Report of 150 Cases. W. A. Boice and W. H. Stenstrom.—p. 234.
*Saddle Block Anesthesia in Obstetrics: Report of 500 Cases. L. R. Pearlman.—p. 242.
Carcinoma of Prostate with Posterior Extension into the Rectum: Report of 3 Cases. J. L. Montgomery.—p. 245.
Ligation of the Common Iliac Vein for Repeated Pulmonary Embolism: Case Report. J. M. Bruner.—p. 248.
Unusual Case of Nontropical Sprue Which Responded to Folic Acid. P. W. Montgomery and W. M. Fowler.—p. 250.

Saddle Block Anesthesia in Obstetrics.—Pearlman says that since Parmley and Adriani in 1946 reported observations on 100 obstetric patients in whom they employed saddle block anesthesia with dibucaine (nupercaine®), he had used this method in 500 cases, using essentially the same technic. In this series there were 196 primigravidas and 304 multigravidas. Four hundred and sixty-two had operative deliveries, and 38 had spontaneous deliveries. The obstetricians conducting this series with the author usually advocated routine outlet forceps; hence the low incidence of spontaneous deliveries. Saddle block anesthesia increases the incidence of operative vaginal deliveries, but this is not considered a contraindication to its use in competent hands. The procedure has an advantage over inhalation anesthesia in patients with cardiac disorders, toxemias and disease of the upper part of the respiratory tract. The primary disadvantage was the occurrence of postspinal headache. The method proved advantageous to the patient, the infant, the delivery room personnel and the obstetrician. It is a safe and simple procedure with minimal complications in the hands of competent anesthesiologists.

Journal of Allergy, St. Louis

20:159-228 (May) 1949

Experimental Sensitization with Food Dyes and Cross-Sensitization to Paraphenylenediamine. R. L. Mayer.—p. 159.
Fixed Anaphylactic Sensitization: Bromsulphalein Studies. J. J. Sievers, G. R. Morey and M. Samter.—p. 167.
Chemical Isolation and Biologic Assay of Extracellular Antigenic Fractions from Pathogenic Fungi. E. L. Keeney and N. Eriksen. —p. 172.
Studies of Capillary Permeability in Allergy with Dermofluorometer. L. H. Criep and M. I. Levine.—p. 185.
Sensitivity to Edible Vegetable Oils. K. D. Figley.—p. 198.
Statistical Study of Emotional Component in Allergy: Its Relationship to Age and Sex Distribution of 5,583 Patients. M. B. Cohen and L. E. Abram.—p. 207.
Transitory Pulmonary Infiltrations (Loeffler's Syndrome) with Case Report. A. M. Tucker.—p. 211.
Vaccinial Infection in Children with Atopic Dermatitis. J. H. Fries and S. Borne.—p. 222.

Journal of Gerontology, Springfield, Ill.

4:90-183 (April) 1949. **Partial Index**

Medical Approach to Problems of Aging. R. A. Moore.—p. 90.
Direct Effect of Steroids on Senile Human Skin: Preliminary Report. J. W. Goldzieher.—p. 104.
Correlation Between Clinical Appearance of Skin and Skin Lipid Secretion in Middle-Age and Old Individuals. S. A. Kvorning and E. Kirk. —p. 113.
Constipation in the Aged, Attempts at Therapy. H. Sorter, M. Berg and H. Necheles.—p. 121.
Serum Protein-Bound Iodine and Age. W. B. Kountz, M. Chieffi and E. Kirk.—p. 132.
Distinctive Features of Coronary Disease in the Aged. E. P. Boas. —p. 136.

Journal of International College of Surgeons, Chicago

12:207-400 (May-June) 1949. **Partial Index**

Thyroiditis: Diagnosis and Treatment. J. Hertz.—p. 211.
*Evaluation of Present Treatment of Hyperthyroidism. J. deJ. Pemberton, S. F. Haines and F. R. Keating Jr.—p. 223.
Present Status of Treatment of Hyperthyroidism. G. Crile Jr.—p. 229.
Malignancy of Thyroid Gland. A. S. Jackson.—p. 234.
Thyrotoxicosis Without Elevated Basal Metabolism. V. E. Chesky and C. R. Schmidt.—p. 238.
Relation of Metabolism to Thyroid Surgery. M. N. Foote.—p. 245.
Radioiodine in Thyroid Disease. G. M. Curtis and M. B. Fertman. —p. 254.
Thyroidectomy—Prevention and Treatment of Complications. D. U. McGregor.—p. 261.
Important Anatomical Structures in Thyroidectomy. M. Nordland. —p. 271.
Thyroid Anesthesia. T. C. Davison and A. H. Letton.—p. 281.
Progress in Dealing with Vocal Cord Paralyses. B. T. King.—p. 288.
Treatment of Hyperthyroidism, Especially with Radioactive Iodine. E. M. Chapman.—p. 301.
Pre- and Postoperative Care of Patient with Hyperthyroidism. W. R. Lovelace.—p. 305.
Preoperative and Postoperative Treatment in Hyperthyroidism. L. Seed. —p. 310.
*Surgical Management of Chronic Ulcerative Colitis. H. E. Bacon and G. D. Vaughan.—p. 320.
Radical Amputation for Cancer of Breast With and Without Irradiation: Statistical Study. C. C. Kennedy.—p. 324.

Present Treatment of Hyperthyroidism.—Pemberton and co-workers report 789 consecutive cases of hyperthyroidism in which thyroidectomy was performed at the Mayo Clinic up to Jan. 1, 1948. Four hundred and fifty-five patients had toxic diffuse goiter. There were no deaths in this group. Of 334 patients who had adenomatous goiter associated with hyperthyroidism, 2 died. The mortality rate for both groups, comprising all the cases of hyperthyroidism, was only 0.25 per cent. Subtotal thyroidectomy was performed as a single stage procedure in all the cases of toxic diffuse goiter and in all but 3 of the cases of adenomatous goiter. The authors feel that thyroidectomy after administration of either strong iodine (Lugol's) solution alone or strong iodine solution and propylthiouracil preparation is the most conservative method of treatment for the majority of patients with toxic diffuse goiter. The short time needed for this method of treatment, the speed of control of hyperthyroidism, the high incidence of permanence of control and the low incidence of morbidity and death make surgical treatment a method which must still be considered in most cases of hyperthyroidism and which will be the treatment of choice in many instances. Propylthiouracil is a valuable drug for the control of hyperthyroidism, but, because of its potential toxic side effects and because of the high incidence of relapses after its withdrawal, employment of this agent as the method of choice in the total treatment of toxic diffuse goiter has not been established. As a means of preparing the patient for operation, medication with iodine has proved adequate except for a small group of patients whose operative risk is high because of severity of hyperthyroidism or because of complications. For this group, treatment with propylthiouracil has been found to be valuable as an additional measure of preparation. The low surgical mortality rate reported herein may be ascribed to these methods of preoperative treatment. Treatment with radioiodine proved to be an effective means of control in many cases of hyperthyroidism. Until greater accuracy in gaging the dose is established and until the potential radiation hazard in the treatment of hyperthyroidism with radioiodine is known, the employment of this drug should be limited to patients for whom thyroidectomy is regarded as unduly hazardous.

Surgical Management of Chronic Ulcerative Colitis.—Bacon and Vaughan customarily prescribe a rigid medical regimen for their patients with chronic ulcerative colitis, which includes individualized diet, adequate vitamins, transfusions of blood and plasma with regular frequency and in small amounts, parenteral administration of protein derivatives and sulfathalidine® alternating with neoprontosil.® Amebicides, such as emetine hydrochloride, may be employed even though repeated mucosal scrapings and stool specimens do not show the parasite. Where severe exacerbations and remissions of the disease have occurred on two or three occasions, surgical intervention is indicated. Ordinarily a preliminary ileostomy is established and subsequent colectomy performed, provided the case conforms to its indications, the patient presenting signs and symptoms of persistent sepsis, extensive scarring of the bowel, fibrous stricture, pseudopolypoid and malignant degeneration and arthritic change. In the authors' experience with 19 ileostomies, performed either as an emergency or elective procedure, the mortality rate was 21 per cent. There were twelve colectomies, either total or partial, without death; with the exception of 1 case, removal of the rectum has been required as the final step.

Journal of Investigative Dermatology, Baltimore

12:255-316 (May) 1949

Tickbite Granuloma. N. Tobias.—p. 255.
Reaction of Psoriasis Following Use of Gamma Globulin. G. H. Klinck Jr.—p. 261.
*Effect of Certain Hyperkinemics on Blood Flow Through Skin. K. Lange and D. Weiner.—p. 263.
Cutaneous Cryptococcosis: Survey of Cryptococcus on Normal and Pathologic Skin. H. G. Ravits.—p. 271.
Additional Studies on Psoriasis in Tropics and in Starvation Camps. R. D. G. Ph. Simons.—p. 285.
Species of Aloe (Other Than Aloe Vera) in Treatment of Roentgen Dermatitis. L. J. A. Loewenthal.—p. 295.
Contact Dermatitis Due to Procaine: Common Occupational Disease of Dentists. E. L. Laden and D. A. Wallace.—p. 299.
Measurement of Pain Threshold and Superficial Hyperalgesia in Diseases of Skin. C. B. Potelunas, M. D. Meixner and J. D. Hardy. —p. 307.

Effect of Hyperkinemic Agents on Blood Flow Through Skin.—Lange and Weiner say that although the public is making wide use of skin-irritating ointments and liniments because of their rubefacient qualities, the members of the medical profession have largely disregarded these agents because of the absence of concrete evidence of therapeutic value. The authors developed a method for the evaluation of the effectiveness of different preparations by estimating the increase in blood flow. Other investigators had demonstrated that some rubefacients, as mustard and turpentine, do not produce an increase in skin temperature in spite of the production of intense redness of the skin. The vasodilation in these cases seems to be mainly venous in character, thus not producing an increase in blood flow through the area. The term rubefacients is therefore ambiguous. Passive and active hyperemia are both included in it, although physiologically and therapeutically there is a great difference between the two. The authors suggest the use of the term hyperkinemics for preparations which increase the blood flow through an area. They list the composition of six counter-irritants which they tested on the arms of 35 volunteers to determine the ability of the agents to produce an increase in skin temperature as measured by thermocouples. The products vary in ability to increase blood flow. Three preparations failed almost completely to increase the skin temperature; one even reduced the skin temperature slightly. One was only slightly effective. The other three preparations produced an increase in temperature but in different degrees. One preparation was highly effective but decreased in effectiveness toward the end of the test period of four and one-half hours. Another preparation was exceedingly effective as well as long lasting. The sensitivity to hyperkinemic agents varies greatly in different persons, but the general pattern of reaction to the individual preparations is rather constant in different persons. The effect of a superficially applied hyperkinemic agent can extend to the depth of 2.5 cm. below the surface of the skin as measured by thermoneedles. The authors believe that hyperkinemic agents can be of real therapeutic value by producing a considerable increase in skin temperature and capillary permeability, leading to a better supply of blood for an area and to the faster removal of metabolites and toxins.

New England Journal of Medicine, Boston

240:747-786 (May 12) 1949

Variant of Hofstätter-Cullen Sign in Intra-Abdominal Hemorrhage from Ectopic Pregnancy with Note on Mechanism of Its Production. J. Fallon and J. J. Manning.—p. 747.
Effect of Oral Therapy with Cobaltous Chloride on Blood of Patients Suffering with Chronic Suppurative Infection. J. C. Robinson, G. W. James III and R. M. Kark.—p. 749.
Erythropoietic Effect of Cobalt in Patients With or Without Anemia. L. Berk, J. H. Burchenal and W. B. Castle.—p. 754.
Methods of Induction in Pediatric Anesthesia. R. M. Smith.—p. 761.
Oral Surgery (Concluded). K. H. Thoma.—p. 765.
Adenocarcinoma (Low Grade) of Ampulla of Vater.—p. 773.
Carcinoma of Ascending Colon, with Metastases to Liver, Regional Lymph Nodes, Lungs and Kidney. Arteriosclerotic Aneurysm of Abdominal Aorta, Wired.—p. 776.

Ohio State Medical Journal, Columbus

45:429-536 (May) 1949

Pyarthrosis in Infancy—Report of Case with Review of Literature. J. V. Greenebaum, J. A. Freiberg and E. L. Saenger.—p. 453.
Myotonia Congenita (Thomsen's Disease): Report of Case. C. R. Freeble Jr. and J. M. Rini.—p. 459.
Aureomycin in Infections of Urinary Tract. R. F. Jukes.—p. 460.
Some Fundamentals in Water and Electrolyte Balance. W. G. Maddock. —p. 462.
Rupture of Appendiceal Stump Three Months After Uneventful Appendectomy with Repair and Recovery. L. O. Baumgardner.—p. 467.

Philippine Medical Association Journal, Manila

25:159-210 (April) 1949

Surgical Problems in Urinary Lithiasis. L. F. Torres Jr.—p. 159.
Public Health Approach to Our Nutrition Problems. J. Salcedo Jr. —p. 165.
Glaucoma Problem in Philippines. G. de Ocampo.—p. 173.
Present Concepts, Practices, and Trends in Public Health Administration in the United States. C. R. Pascual.—p. 181.
Observations on Hookworm Anemia in the North General Hospital. J. R. Cruz, A. Guytingco and W. R. Kasilag.—p. 191.
Dermoid Cyst of Urinary Bladder with Calculus: Case Report. A. A. Galvez.—p. 195.
Management of Sterility in Bontoc Hospital. F. V. D'Bayan.—p. 197.

Proc. of Staff Meet. of Mayo Clinic, Rochester, Minn.

24:277-308 (May 25) 1949

*Effects of Adrenal Cortical Hormone 17-Hydroxy-11-Dehydrocorticosterone (Compound E) on Acute Phase of Rheumatic Fever: Preliminary Report. P. S. Hench, C. H. Slocumb, A. R. Barnes and others.—p. 277.
Observations on Hormone of Adrenal Cortex Designated Compound E. E. C. Kendall.—p. 298.
Oral Use of Aureomycin in Treatment of Late Cutaneous Syphilis. P. A. O'Leary, R. R. Kierland and W. E. Herrell.—p. 302.
Tuberculous Anal Fistula with Subsequent Development of Pulmonary Tuberculosis: Report of Case. R. J. Jackman and N. D. Smith. —p. 307.

Effects of the Adrenal Cortical Hormone (Compound E) on Acute Rheumatic Fever.—Hench and associates administered the adrenal cortical hormone, 17-hydroxy-11-dehydrocorticosterone (compound E, Kendall) to 3 adolescent patients with acute rheumatic fever. Two of the patients were in the first attack of acute rheumatic fever; the third probably was in a recurrent attack. Compound E acetate was administered intragluteally as a suspension in a sodium chloride solution of finely ground crystals in a concentration of 25 mg. per cubic centimeter. The total daily doses generally were 200 mg. (100 mg. given twice a day) for the first several days, and thereafter 100 mg. (50 mg. given twice a day) for a few days more. Prior to admission to the hospital the patients had received for a few days sulfonamide compounds, penicillin or salicylates, or a combination of these, generally without significant effect. During the few days prior to the administration of the compound, while preliminary clinical, biochemical and electrocardiographic studies were being made, the patients received either no medicament or a few small doses of codeine, which likewise was discontinued before use of the compound was begun. In each of the 3 patients treatment with the agent was followed by the rapid disappearance not only of the fever, tachycardia and polyarthritis but also of the elevated sedimentation rates and abnormal electrocardiographic changes. In view of the beneficial effect of the endocrine substance on the skeletal muscles and fibrous tissues of patients with rheumatoid arthritis, it may be hoped that the compound will exert a similar effect on the cardiac muscles and fibrous

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valves in rheumatic fever, an effect which salicylates cannot produce. Even though the effects observed in this study are encouraging, it must be emphasized that these patients will have to be observed for many months before it can be determined whether or not treatment with the compound has prevented or lessened the chronic organic injury to the heart valves or myocardium. No supplies of compound E are expected for treatment or additional research until sometime in 1950 at the earliest, when supplies still will be exceedingly small.

Public Health Reports, Washington, D. C.

64:657-684 (May 27) 1949

Birth of Community Mental Health Clinic. E. Davens and P. Lemkau. —p. 657.
Effects of DDT Dusting on Domestic Rats Under Colony and Field Conditions. J. E. Dent, H. B. Morlan and E. L. Hill.—p. 666.
Avirulent Isolate of Salmonella Typhosa 58 (Panama Carrier). H. C. Batson, M. Landy and A. Abrams.—p. 671.

64:685-716 (June 3) 1949

Studies of Patients Discharged from Tuberculosis Sanatoria: II. Mortality Rates Associated with Selected Characteristics of the Patient Population. Agnes W. Brewster and R. C. Fletcher.—p. 687.
Characteristics of Commercial X-Ray Screens and Films.—VI. W. W. Van Allen.—p. 706.

64:717-748 (June 10) 1949

Summary of Antimalarial Drugs. W. C. Cooper.—p. 717.
Prevalence of Poliomyelitis in 1948. C. C. Dauer.—p. 733.

Radiology, Syracuse, N. Y.

52:777-932 (June) 1949

Peptic Ulcer: Importance of Films in Diagnosis. F. E. Templeton. —p. 777.
Place of Fluoroscopy in Diagnosis of Peptic Ulcer. M. Harris.—p. 781.
Physical Problems of Fluoroscopy and Spot-Film Radiography. R. H. Morgan.—p. 786.
Gastroscopy in Evaluation of Patients with Peptic Ulcer. M. E. Dailey. —p. 796.
Peptic Ulcer: Modern Criteria for Diagnosis and Attempted Diagnosis of Healing. D. L. Wilbur and M. S. M. Watts.—p. 800.
Evaluation of Effects of Radiation on Non-Malignant Lesions of Nasopharynx. M. H. Cutler, R. E. Marcus and F. L. Lederer.—p. 816.
*Endocrine Factors in Hypertension; Treatment by Roentgen Irradiation of Pituitary and Adrenal Regions. J. H. Hutton, J. T. Case, E. C. Olson, W. W. Furey, S. Fahlstrom, W. L. Culpepper and E. E. Madden.—p. 819.
Rapid Quantitative Scale Method for Estimating the Percentage Concentration of Priodax in Gallbladder. M. H. Poppel and Celia Bercow. —p. 829.
Sensitometry of Roentgenographic Films and Screens. R. H. Morgan and W. W. Van Allen.—p. 832.
Relationships Between Chemical and Biologic Effects of Ionizing Radiations. R. E. Zirkle.—p. 846.
Structural Differences in Bone Matrix Associated with Metabolized Radium. F. E. Hoecker and P. G. Roofe.—p. 856.
Individualizing Tumor Dose in Carcinoma of Uterine Cervix by Means of Simple Caliper. J. B. Jarvis.—p. 866.
Effects of Continuous Radiation on Chick Embryos and Developing Chicks: II. Bone Marrow, Lymphoid Tissue, and Peripheral Blood. S. Warren and F. J. Dixon.—p. 869.

Endocrine Factors in Hypertension.—Hutton and associates report 795 patients with hypertension; 486 of these were seen by one or more of the group of the authors, and 309 were reported to them by other workers. They had also seen 42 patients with both hypertension and diabetes, but these are not included in this report. They arrive at the conclusion that at least a small proportion of cases of hypertension have an endocrine background and that every patient who has this disorder should have an endocrine study. The pituitary and adrenals seem to be most often involved. Roentgen irradiation was directed to these regions in an attempt to control symptoms and lower the blood pressure. The authors have themselves treated 413 patients with improvement (a lowering of blood pressure of at least 30 mm. systolic and 10 mm. diastolic, with symptomatic relief) in 70 per cent of those receiving adequate treatment. Factors which have been found most satisfactory were 120 kilovolts, 3 to 5 milliamperes, 3.0 mm. of aluminum and 50 cm. target skin distance. Roentgen rays were directed to three areas at each sitting—two pituitary portals, each 10 by 10 cm., and a 15 by 15 cm. portal over the adrenals —about 50 r, measured in air, being given to each portal. The blood pressure was checked repeatedly after treatment, and roentgen therapy was repeated only when there was no favorable response to the first treatment or there was a recurrence of symptoms or a rise in pressure. As many as six treatments may be given in a series at weekly intervals, and the series may be repeated as needed. Eventually, the treatment ceases to be effective (after six to eight years in the authors' experience). No evidence of roentgen damage has been observed following this treatment. In 2 cases autopsy revealed no injury to the pituitary, adrenals or other tissues or organs.

Review of Gastroenterology, New York

16:369-450 (May) 1949

Diabetes Mellitus and Gastrointestinal Disease. H. O. Mosenthal. —p. 381.
Tuberculosis in Gastrointestinal Cases. C. Muschenheim.—p. 384.
Psychosomatic Involvements in Gastrointestinal Cases. R. A. Gerber. —p. 387.
Cardiac Manifestations of Gastrointestinal Disease. H. E. B. Pardee. —p. 395.
*Modus Operandi of Carminatives: Therapeutic Value of Garlic in Functional Gastrointestinal Disorders. F. Damrau and E. A. Ferguson.—p. 411.

Garlic in Functional Gastrointestinal Disorders.—Damrau and Ferguson point out that although the value of garlic as a carminative has been recognized since ancient times, few scientific studies on the modus operandi of carminatives have been published. Cushny in 1940 had suggested that the carminative oils cause a sensation of warmth on passing into the stomach; this is accompanied by a sense of well-being and comfort, and any feeling of distention after meals is relieved. This is often attended by eructation of quantities of gas. The authors believe that their studies have confirmed Cushny's theory. A preliminary roentgenographic study was undertaken to determine the effect of dehydrated garlic on gastrointestinal motility by comparison of the opaque residues with and without the garlic. The authors describe clinical studies and present reports which are representative of therapeutic results with complete symptomatic relief obtained with dehydrated garlic tablets. In this form garlic is highly effective for relief of heaviness after eating (epigastric and abdominal distress), belching, flatulence, gas colic and nausea. Satisfactory therapeutic results were obtained in cases of flatulent dyspepsia, nervous dyspepsia and other gastric neuroses. Roentgenographically a comparison of films of patients with and without the medication demonstrated that dehydrated garlic has a sedative action on the stomach and intestines, relaxes spasm, retards hyperperistalsis and disperses accumulations of gas. It is believed that these studies explain the carminative action of garlic as due to unidentified principles which have been designated as gastroenteric allichalone (allium, garlic; chalone, to relax). Since dehydrated garlic tablets are safe for long-continued use, they may be prescribed in a wide variety of functional disturbances of the stomach and intestines.

Surgery, Gynecology and Obstetrics, Chicago

88:689-806 (June) 1949

*Methods for Preservation and Transplantation of Arterial Grafts: Observations on Arterial Grafts in Dogs: Report of Transplantation of Preserved Arterial Grafts in 9 Human Cases. R. E. Gross, A. H. Bill Jr. and E. C. Peirce.—p. 689.
Testosterone Propionate in Treatment of Advanced Carcinoma of Breast. J. R. Watson and G. H. Fetterman.—p. 702.
Study of Peripheral Vascular Disease with Radioactive Isotopes: Part II. F. W. Cooper Jr., D. C. Elkin, P. C. Shea Jr. and E. W. Dennis. —p. 711.
Suture of Peripheral Nerves: Factors Affecting Prognosis. J. W. Kirklin, F. Murphey and J. Berkson.—p. 719.
*Stratification of Bile in Gall Bladder and Cholelithiasis. B. A. Campbell and A. C. Burton.—p. 731.
Vascular Changes of Skin in Pregnancy: Vascular Spiders and Palmar Erythema. W. Bean, R. Cogswell, M. Dexter and J. F. Embick. —p. 739.
Thoracic Complications of Amebiasis. R. R. Shaw.—p. 753.
Carcinoma of Nasopharynx: Report of 150 Cases. M. W. Simmons and I. M. Ariel.—p. 763.
Uncommon Tumors of Salivary Glands. M. C. Wheelock and T. J. Madden.—p. 776.
*Stimulation of Ovarian Function and Induction of Pregnancy Through Intravaginal Implantation of Estrogen Pellets. B. Zondek and S. Rosin. —p. 783.

Preservation and Transplantation of Arterial Grafts.—Gross and associates describe their work on quick-freezing grafts of the blood vessels and storing them at 70 C. In a second series they investigated the feasibility of preservation

and use of heterografts. In the third series they investigated the preservation and use of homografts, storing the grafts at temperatures just above freezing in appropriate mediums. Segments of large arteries, or the aorta, were accumulated from human subjects who had died in automobile accidents. The vessels were removed within a few hours after death of the person, and in no instance was the lapse greater than five hours. The acquired segments of vessel—iliac, carotid, subclavian and aortic—were preserved in individual flasks and covered with balanced saline solution. These flasks were then stored at 1 to 4 C. Eight of these human grafts were implanted in patients who had a cyanotic type of congenital heart disease and in whom it was desirable to make some sort of a shunt of blood from the aorta or one of its branches into the pulmonary artery. Ordinarily it is possible to bring such vessels together for a direct anastomosis by the Blalock or the Potts technic, but sometimes such shunts cannot be made without excessive tension and in such a case a graft will allow arterial junctions to be made without tension and will give a result which is better than that which would have been obtained from a poor, direct anastomosis. These same general principles will probably also hold true for treatment of coarctation of the aorta. The use of an interposed graft may prove to be the most satisfactory method of reestablishing an aortic pathway of normal size. The validity of this statement is suggested by the result obtained in a case wherein a graft was used to repair an aortic defect after excision of a coarctation. Histories of 9 cases are presented. The authors feel that the promising results justify further trial and study.

Stratification of Bile in Gallbladder.—Campbell and Burton point out that, although the literature on the composition of bile in the gallbladder and on the concentrating action of that organ implies that its contents at any one time are homogenous, there has been roentgenologic evidence that a high degree of inhomogeneity of bile may exist in the human gallbladder in normal subjects as well as in patients with cholecystitis. Samples of bile withdrawn at different levels in dependent gallbladders of cats, cattle and some human beings were examined as to their specific gravity, concentration of pigments, viscosity and hydrogen ion concentration. A lack of homogeneity from top to bottom was the rule rather than the exception. Of 65 gallbladders of normal cattle, 60 per cent showed an increase in specific gravity, from top to bottom, of 0.005 to 0.023 unit. Changes in hydrogen ion concentration and in color index closely paralleled the changes in specific gravity. Changes in viscosity were not well correlated. The changes were often so abrupt as to show the existence of two or more distinct stratums. Neither the process of diffusion for as long as a week nor considerable mechanical agitation was able to mix these stratums. In 2 of the 5 human cases, cholesterol stones were floating at the level of the interspace between two stratums. A theory of the formation of such strata in terms of incomplete emptying of a gallbladder is suggested. The possibility is discussed that persistent stratification of bile may favor the precipitation of cholesterol stones.

Stimulation of Ovarian Function by Intravaginal Implantation of Estrogen Pellets.—Zondek and Rozin demonstrated experimentally that the activity of estrogenic hormone was greatly increased when the hormone was injected near its target organ, i. e., in the vaginal submucosa. They present case histories illustrating the effect of intravaginal implantation of estrogen pellets on primary and secondary amenorrhea. They stress that intravaginal implantation possesses the following advantages over subcutaneous administration: The effect is more constant; it is about five times as intense, and the tablets are seldom expelled. The effects of implantation are manifested in improved vascularization of the vagina, changes in the vaginal epithelium (cornified cells), uterine growth, proliferation of the uterine mucosa, uterine bleeding and enlargement of the breasts. The average interval between implantation and the onset of bleeding is seventy-eight days. The cycle may be reinitiated by a single intravaginal implantation, whereby the amenorrhea is relieved. Alternatively, several cyclic bleedings may occur, which are again followed by amenorrhea, requiring a second or third implantation. A second implantation is also indicated if no bleeding occurs within three to four months of the initial implantation. Doses ranged between 25 and 50 mg. of estrone or estradiol. Undesirable secondary effects, such as an excessively intense peripheral reaction involving glandular cystic hyperplasia of the uterine mucosa, sometimes accompanied with severe bleeding, may result. Although this may require treatment with progesterone or even curettage, this accessory effect should not discourage repetition of implantation of a smaller dose. The authors have employed their method of intravaginal implantation in 54 married women with amenorrhea of many years' duration, accompanied with severe secondary changes of the genital organs. Bleeding was induced in 84 per cent of the amenorrheic patients. In 8 of the treated patients, not only was the amenorrhea cured but pregnancy ensued.

Tennessee State Medical Assn. Journal, Nashville

42:147-182 (May) 1949

Menace of Socialized Medicine: Call to Action for Personal Privilege and Professional Liberty. H. W. Qualls.—p. 147.
Observations on Treatment of Benign Gastric Ulcer: Report of Case of Benign Gastric Ulcer Treated Medically. R. A. Cate.—p. 152.
Report of Fatal Case of Post-Vaccinal (Allergic) Encephalitis. J. T. Moore.—p. 157.

42:183-222 (June) 1949

Shelby County Doctors vs. Socialized Medicine. W. C. Chenay.—p. 183.
Early Diagnosis and Treatment of Carcinoma of Prostate Gland. P. Niceley.—p. 186.
Surgical Treatment of Malignant Lesions of Gastrointestinal Tract. J. H. Forsee and R. M. Hardaway.—p. 195.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

57:181-220 (May) 1949

Pregnancy and Cerebrovascular Complications: Report of 7 Cases. D. W. deCarle.—p. 181.
*Oral Basal Temperatures and Diagnosis of Pregnancy. H. L. Stewart Jr.—p. 192.
Two Case Reports of Sympus. J. G. Busby.—p. 201.
Surgical Treatment of Carcinoma of Esophagus and Cardia. H. L. Thompson.—p. 205.
Carcinoma of Prostate. O. J. Jensen Jr. and J. A. Moore.—p. 210.

Oral Basal Temperatures in Pregnancy.—Stewart states that the oral basal temperatures in women with a normal menstrual cycle show a biphasic curve characterized by a lower preovulatory (estrin) phase followed by an elevated postovulatory phase (progestin). Oral basal temperature curves in the event of pregnancy show a continuation of the postovulatory elevation. There is a gradual drop in this level in most cases during the third month to a level of 98 F., with wide daily fluctuations. This gradual fall continues, so that the preovulatory level is reached by the fourth or fifth month of gestation and remains until delivery. Because of the gradual fall in basal temperatures during pregnancy, the oral basal temperature is of valuable diagnostic aid only during the first trimester. The accuracy of the test is as great as the accepted biologic tests for pregnancy, but it must be used in conjunction with physical examinations and clinical judgment. Basal temperature should be taken for at least seven consecutive days. Oral basal temperatures of 98.2 F. or above for seven consecutive days in the absence of an abnormal temperature rise at other periods of the day favors a clinical diagnosis of pregnancy. A temperature of 97.6 F. or below is against a diagnosis of pregnancy. The accuracy of the test in their series of 425 cases was 97.5 for cases with positive results and 99 per cent for the cases with negative results.

West Virginia Medical Journal, Charleston

45:101-124 (May) 1949

Curare in Anesthesia. J. F. Morris.—p. 101.
Use of Base-In Prisms in Treatment of School Myopia. O. B. Nugent.—p. 103.
Management of Compound Fracture. W. Bronaugh.—p. 106.
Ureterocele with Prolapse Through Urethra: Case Report. A. J. Oliker.—p. 109.

45:125-158 (June) 1949

Anthracosilicosis and Its Symptomatic Treatment. B. Gordon, H. L. Motley, P. A. Theodos and L. P. Lang.—p. 125.
Approach to X-Ray Film. K. J. Myers.—p. 133.
Pathologic Conscious States. E. F. Reaser.—p. 136.
Diagnosis and Management of Complications of Peptic Ulcer. H. Acuff.—p. 141.

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FOREIGN

An asterisk (*) before a title indicates that the article is abstracted. Single case reports and trials of new drugs are usually omitted.

Brain, London

72:1-112 (March) (Part I) 1949

Muscle Spindles in Human Extrinsic Eye Muscles. Sybil Cooper and P. M. Daniel.—p. 1.
Amyotonia Congenita. J. W. A. Turner.—p. 25.
Proprioception and Motor Cortex. E. Gellhorn.—p. 35.
Significance of Tinel's Sign in Peripheral Nerve Injuries. J. R. Napier. —p. 63.
*État Marbré of Thalamus Following Birth Injury. R. M. Norman. —p. 83.
Disorders of Body Image Caused by Lesions of Right Parietal Lobe. M. Roth.—p. 89.

Marble State of Thalamus Following Birth Injury.—Norman reports a case in which "marble state" of the thalamus was present in conjunction with other lesions the result of vascular damage occurring at birth. The patient was a male idiot with cerebral diplegia who died at the age of 5 years and 3 months. The infant was born prematurely and weighed 5 pounds (2,495 Gm.). There was a history of severe asphyxia at the time of birth and twitchings of the hands and face observed on the fifth day of life. He appeared to be blind and had pale optic disks. Although a slight improvement was noted in the infant's physical progress, he was never able to stand or sit up because of a symmetric spastic paresis which affected the legs more than the arms. There were occasional seizures of a tonic type. Examination of the brain revealed a bilateral "état marbré" (marble state) of the thalamus and conspicuous areas of softening in each centrum semiovale adjacent to the lateral ventricles. The pyramidal tracts at medullary level were of the size and degree of myelination approximately appropriate for a newborn infant. The optic radiations and the external geniculate bodies showed partial destruction and there was an apparent increase in the neuroglial fibrils of the optic nerves and optic papilla. It was concluded that the lesions of the thalami and central white matter, which lay within the drainage area of the great vein of Galen, were late neuropathologic sequelae of birth injury.

British Journal of Ophthalmology, London

33:329-396 (June) 1949. **Partial Index**

Diseases of Eye in Relation to Dental Surgery. J. H. Doggart.—p. 338.
Concentration in Aqueous of Various Sulphonamides After Systemic Administration. A. Sorsby.—p. 347.
Significance of Ocular Complications Following Vaccination. E. Rosen. —p. 358.
Treatment of Hypopyon Ulcer of the Cornea. E. H. Cameron.—p. 368.
Modern Views of Surgery of the Cornea. M. S. Bey.—p. 372.
Injection of Retinal Vascular System in Enucleated Eyes. I. C. Michaelson and H. F. Steedman.—p. 376.
*Lacrimal Strip and Precorneal Film in Cases of Sjögren's Syndrome. M. Klein.—p. 387.

Precorneal Film in Sjögren's Syndrome.—Klein quotes Wolff's suggestion that the precorneal film has a triple structure, consisting of a deep mucoid layer, an intermediate lacrimal watery layer and an oily layer. The mucous layer derives from the goblet cells of the conjunctiva, the watery layer from the lacrimal glands and the oily layer from the tarsal glands. The lacrimal layer is responsible for the brightness of the corneal surface. An accumulation of this watery layer, the marginal strips, can be seen in the gutter formed by the lid margins. Sjögren has pointed out that in keratoconjunctivitis sicca the conjunctiva and cornea lose their brightness. Klein studied the lacrimal strip in several patients with this disorder. Although Schirmer's test indicated considerable reduction of lacrimal secretion in all the cases studied, the marginal strips of tear fluid were present. The precorneal film lacked the easy-flowing, flexible appearance of the normal film and was rather viscous. The precorneal film seemed to be composed of the usual three elements, but the mucoid component apparently predominated over the lacrimal film. Whether this was due to diminished lacrimal secretion alone, or the composition of the tear fluid was altered as well, needs further investigation. According to Ridley, the protein content of the tears is reduced in Sjögren's syndrome. This may be significant, as the surface tension of the tears is influenced by the protein content.

British Journal of Venereal Diseases, London

25:45-100 (June) 1949

Medical Conduct of a Brothel. F. R. Bettley.—p. 56.
Modern Interpretations of Serum Tests. I. N. Orpwood Price.—p. 67.
Syphilis Antigen from Soya Bean. J. S. Stevenson.—p. 78.
Gonorrhea and Sulphonamides. E. M. C. Dunlop.—p. 81.
Advertisements on Treatment of Venereal Disease and Social History of Venereal Disease. A. Fessler.—p. 84.

British Medical Journal, London

1:969-1018 (June 4) 1949

Surgical Aspects of Meningitis. H. Cairns.—p. 969.
Ayerza's Disease, Silicosis and Pulmonary Bilharziasis. H. Tidy. —p. 977.
*Incidence of Cancer of Uterine Cervix. R. G. Maliphant.—p. 978.
Psychiatric Patients and Disabled Persons (Employment) Act. A. Harris and M. A. Lane.—p. 982.
Phyto-Photo-Dermatitis. H. E. Bellringer.—p. 984.
Prognosis and Treatment of Sprue in India. K. D. Keele.—p. 986.

Cancer of Uterine Cervix.—Maliphant attempted to ascertain the incidence of uterine cancer together with the relative probabilities of the disease developing in the various age, social state and parity groups of the population. Studies were made on 1,200 women with cancer of the uterine cervix, and certain of their characteristics were compared with the distribution of the same characteristics in the community. Cancer of the cervix is a disease of middle life, but the risk of contracting the disease continues to rise until the age of 60. Beyond this age the risk shows a steady decline: while the female population is decreasing, the incidence of cervical cancer is decreasing at a faster rate. The cancer hazard is increased by marriage irrespective of childbearing, but the disease shows a predilection for women who have borne one or more children. Its occurrence seems to be independent of the nature of the confinement, but each pregnancy adds slightly to the risk that cancer may develop in later years. In women of cancer age (35 to 64 years) the probability of the disease developing in any one year seems to range from 1 in 21,000 single women, through 1 in 6,500 in childless married women to 1 in 1,500 in parous women. There is little prospect of effecting any appreciable reduction in the incidence of the disease by prophylactic measures. The pressing need is diagnosis at a stage when cure is not only possible but probable by existing therapeutic methods. The role of periodic health examinations in the detection of symptomless cervical cancer is discussed.

Journal of Neurol., Neurosurg. & Psychiatry, London

12:81-166 (May) 1949

"Integration, Then and Now." E. G. T. Liddell.—p. 81.
Fate of Intraneural Injection as Demonstrated by Use of Radio-Active Phosphorus. J. B. Brierley and E. J. Field.—p. 86.
Metachromatic Bodies in Brain. M. C. Smith.—p. 100.
Electrical Activity of Neostriatum, Paleostriatum and Neighboring Structures in Parkinsonism and Hemiballismus. R. Meyers, R. Hayne and J. Knott.—p. 111.
Duration and Form of Action Potential in Normal Human Muscle. I. Petersén and E. Kugelberg.—p. 124.
Electromyography in Muscular Dystrophies: Differentiation Between Dystrophies and Chronic Lower Motor Neurone Lesions. E. Kugelberg. —p. 129.
Anterior Inferior Cerebellar Artery: Its Variations, Pontine Distribution, and Significance in Surgery of Cerebello-Pontine Angle Tumors. W. J. Atkinson.—p. 137.
*Some Unusual Complications of Herpes Zoster. C. W. M. Whitty and A. M. Cooke.—p. 152.
Pinealoma with Solitary Spinal Metastasis. F. R. Magarey and H. R. I. Wolfe.—p. 155.
Hysteria in One of a Pair of Identical Twins. J. H. Paterson.—p. 160.

Complications of Herpes Zoster.—Whitty and Cooke describe 3 patients with herpes zoster, all of whom showed myelitis as a complication of the infection. Two had recurrent attacks of zoster in different skin zones over a period of two or three months, thus showing a difference from the usual behavior of the zoster virus, which confers lifelong immunity by a single attack. The authors consider the possibility that these were really cases of herpes simplex. The third patient had a zoster infection precipitated by injury to the fifth nerve. This case exemplifies a "symptomatic" zoster, secondary to peripheral trauma of the fifth cranial nerve. This is not uncommon after involvement of the first sensory neurone, especially at root level, by secondary neoplasm or tuberculous granuloma.

Its cause is generally supposed to be the zoster virus, trauma being only a precipitating factor. Such a view is supported in this case by the sequel of midbrain encephalitis, inexplicable on a purely traumatic basis.

Lancet, London

1:943-986 (June 4) 1949

Deep Pain Sensibility. J. H. Kellgren.—p. 943.
*Ligation of Common Carotid Artery: Report of 19 Personal Cases. L. Rogers.—p. 949.
Serologic Diagnosis of Human Toxoplasmosis. A. Macdonald.—p. 950.
Replacement Transfusion: Technic in Newborn. J. W. Farquhar and I. C. Lewis.—p. 953.
*Sensitisation of Penicillin-Resistant Pathogens. M. George and K. M. Pandalai.—p. 955.
Anuria Following Ischemia of Renal Cortex. A. Solymoss.—p. 957.
Thyrotoxicosis with Ophthalmoplegia, Myopathy, Wolff-Parkinson-White Syndrome and Pericardial Friction. J. A. Strong.—p. 959.

Ligation of Common Carotid Artery.—According to Rogers, ligation of the carotid artery may produce complications either because of immediate cerebral ischemia or because of delayed embolism or thrombosis. These dangers may to a large extent be avoided by ligating the common rather than the internal carotid artery and by section of the artery rather than tying it in continuity. Division of the artery not only severs the accompanying sympathetic fibers and abolishes spasm and prevents recanalization but also reduces the liability to delayed embolism, since the distal retracted part of the artery, which may contain clot at the site of the ligature, is no longer subjected to the pulse wave, as it must be if the artery is tied in continuity. It is therefore important to divide the artery between ligatures. From experiences on 19 patients in whom the author has ligated the common carotid artery without mishap combined with a review of recently published cases he concludes that it is safe to divide the great vessel in the neck. Experience of delayed hemiplegia in 1 case of internal carotid ligation along with a series of cerebral complications and fatalities in recently published cases of ligation of this artery in the neck leads the author to assert that ligation of the internal carotid artery as a primary procedure is dangerous. If it is proposed to ligate the internal carotid in the neck, the common carotid should first be divided and then, some weeks or months later, the internal artery resected. By so doing not only is a collateral circulation encouraged to develop in the circle of Willis but also precautions are taken against delayed complications.

Sensitization of Penicillin-Resistant Pathogens.—George and Pandalai previously reported the difference in susceptibility of certain pathogens to penicillin associated with differences in their metabolic reactions as well as with the presence or absence of certain catalytic systems. They found that magnesium ions can render certain gram-negative penicillin-resistant pathogens, such as Escherichia coli, sensitive to penicillin. Thus clear sensitivity to penicillin seems to depend essentially on a chemical or metabolic factor. To confirm this the authors grew penicillin-sensitive and penicillin-resistant organisms together, isolated them, and ascertained the least amount of penicillin needed to inhibit growth of the strains so isolated. They describe five experiments and stress that the growing of penicillin-sensitive and penicillin-resistant organisms together may cause the penicillin-resistant organisms to become penicillin-sensitive. Growth together for half an hour at 37 C. is in some cases sufficient to induce such sensitivity. The acquired sensitivity lasts a fairly long time. Their earlier postulate that complex catalytic systems are present in pathogens and are responsible for either sensitivity or resistance to penicillin is supported by these results. The factor responsible for the sensitivity to penicillin is a labile constituent of the bacterial cell probably the ribonucleic acid.

Medical Journal of Australia, Sydney

1:633-668 (May 14) 1949. **Partial Index**

Rh Antibody Specificities Found in 115 Human Anti-Rh Serums. R. T. Simmons.—p. 633.
Clinical Value of Liver Function Tests. K. S. Hetzel.—p. 636.
Testing for Rh Factor in Blood of Emergency Patients. V. I. Krieger, S. Weiden and I. O'Loughlin.—p. 646.

Archivum Chirurgicum Neerlandicum, Arnhem

1:1-88 (No. 1) 1949. **Partial Index**

*Pericardiectomy for Constrictive Pericarditis. L. D. Eerland.—p. 6.
Surgical Treatment of Carcinomata of Distal Three Quarters of Thoracic Esophagus and Cardia. J. F. Nuboer.—p. 14.
So-Called Primary Enterococcal Peritonitis. J. Kweekel.—p. 38.
Retropubic Prostatectomy of Terence Millin. J. A. Weijtlandt.—p. 43.
*Androgen-Control Therapy in Carcinoma of Prostate. H. Muller.—p. 77.

Pericardiectomy for Constrictive Pericarditis.—Eerland reports 14 cases in which an operation was performed to correct constrictive pericarditis. Three of the patients died during operative intervention and 1 died two days after the operation. The deaths occurring during operation resulted from piercing of the heart muscle, which apparently had become too brittle. Five patients were cured and 2 were greatly improved. In 1 patient the early results were good, but he could not be traced. Some of those in whom a cure was obtained have survived four, five and six years. Since the ultimate result depends largely on the condition of the heart muscle and the liver, it is important that the pericardiectomy be not unnecessarily delayed. In the presence of tuberculosis operative intervention should be deferred until the symptoms of the activity of the inflammation have disappeared. It is advisable to limit the intervention to partial pericardiectomy above the ventricles. Intratracheal narcosis offers advantages over local anesthesia. In the series described a parasternal extrapleural approach was selected.

Androgen-Control Therapy in Carcinoma of Prostate.—According to Muller androgen-control therapy is based on the premise that prostatic carcinoma can develop or extend further only in the presence of a specific stimulating factor, such as androgenic hormone. Treatment is instituted with the object of inhibiting or abolishing the production of androgen. Roentgen irradiation of the organs governing the production of androgen, orchiectomy and treatment with estrogen have been the methods used to achieve control of androgen and with it the control of prostatic cancer. Recently most investigators have come to the conclusion that a combination of orchiectomy with estrogen therapy will produce the best results. Orchiectomy removes the main source of androgen, while administration of estrogens suppresses the extragonadal production of androgen. From 5 to 10 mg. of diethylstilbestrol is given daily, if the prostatic carcinoma causes no pain. If there are signs of recurrence, the author advises castration together with 1 to 3 mg. of diethylstilbestrol. The same treatment with possibly an increase in the dosage of the endocrine substance is advised if metastases cause pain. In the presence of recurrence, increase in the dose of diethylstilbestrol should be combined with roentgen irradiation. Transurethral resection should be performed if obstructive signs do not yield to other measures. Regular follow-up of the phosphatase level and the sedimentation rate of the patient is necessary so that increase in the dosage of diethylstilbestrol or castration can be decided on early. The main obstacle to the adoption of a definite scheme of treatment is the wide variation in the reactions of different patients.

Deut. Ztschr. f. Verdauungs-& Stoffwechselkr., Leipzig

9:113-160 (April) 1949

Sulfaguanidine in Treatment of Acute and Chronic Diarrheas of Non-Infectious Origins. N. Henning and H. Stadler.—p. 113.
Treatment of Intestinal Tuberculosis with Thiosemicarbazon. F. Kuhlmann and H. Schütt.—p. 124.
*Esophago-Gastro-Enterocolitis (Intestinitis Gravis) with Necrotic Tendencies: High Incidence During Period from 1946 to 1948. K. Kloos.—p. 141.

Esophagogastroenterocolitis (Intestinitis Gravis).—Kloos directs attention to an intestinal disorder which occurred in epidemic proportions during 1946 to 1948 in a part of northern Germany, particularly in the region of the cities Hamburg, Lübeck and Kriel and in Schleswig-Holstein. The author had the opportunity to make necropsy or biopsy studies in 173 cases. Bacteriologic studies were made at the university of Kiel in about 100 cases. The cell structure of the lesions varied greatly. After describing the morphologic aspects and the localization of the lesions, the author discusses the relationship between the clinical aspects and the four stages of the diseases: (1) catarrhal serous enteritis; (2) necrotizing enteritis; (3) ulcerous enteritis, and (4) chronic suppurative granulomatous enteritis. Possible

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complications are listed. The time, cause and frequency of the fatal outcome are discussed in relation to the progress of the disease and to the therapeutic measures employed. When cases of this intestinal disorder were first observed interpretation proved difficult, although the disease has been mentioned for over a hundred years in the world literature under a great variety of terms, such as nonspecific, deep-seated enterogenic inflammation of the intestinal wall; enterocolitis phlegmonosa; necrotic enterocolitis, and enterocolitis submucosa suppurativa. The problem of terminology becomes even more confusing when processes localized in certain sections of the intestine are considered. In this connection the author cites regional or terminal ileitis (Crohn's disease) and similar changes in the large intestine referred to in the French literature as inflammatory tumors and in other reports as inflammatory tumors or stenoses. Since the cause of the disease is not definitely known, and in view of the varied morphologic manifestations, the author suggests the term intestinitis gravis. Observations indicate that the disease is an infectious process, but the author doubts that the bacteria considered by some, such as the anaerobic Bacillus fundiliformis, or an organism of the Welch-Fränkel type, are really the causative organisms. He assumes that a virus or a virus and bacteria may play an etiologic part.

Medicina, Buenos Aires

9:1-84 (Feb.) 1949. **Partial Index**

Sympathectomy in Primary Arterial Hypertension: Late Results. T. Padilla, P. Cossio and J. A. Berreta.—p. 19.
Artificial Aortic Valve: Experiments in Animals. I. Perianes and J. A. Berreta.—p. 29.

Sympathectomy in Arterial Hypertension.—Padilla and collaborators report on bilateral extensive sympathectomy performed on 27 patients with essential arterial hypertension. The patients were observed for several years before the operation and for two to three years after. Frequent examinations were made. The cases were divided into three groups: very favorable, favorable and unfavorable for surgical intervention, according to age, reaction to sedatives, ophthalmoscopic examination and visceral changes. The failure or success of the operation was judged by the survival, the appearance or disappearance of cardiac, renal or cerebral disturbances, changes in the fundus of the eye and the arterial pressure. Three of the patients showed improvement, 11 remained unchanged, 5 grew worse and 8 died as the result of the illness.

Medizinische Klinik, Munich

44:657-688 (May 27) 1949. **Partial Index**

*Experiences with Penicillin in 370 Syphilitic Patients. C. M. Hasselmann.—p. 657.
*Streptomycin for Conservation of Human Milk: Clinical Experiences. F. Linneweh.—p. 666.
Symptomatology of Primary Tuberculous Infection in Adults. R. W. Muller.—p. 671.
Prescription of Digitalis. H. von Hoesslin.—p. 672.

Penicillin in Treatment of Syphilis.—On the basis of observation in 370 cases, Hasselmann believes that penicillin therapy is superior to any other treatment of syphilis, both as to the reliability of cure and the avoidance of untoward secondary effects such as those associated with arsphenamine therapy. The advantages of penicillin therapy should not tempt physicians to commence its administration before the diagnosis of syphilis has been definitely established or to give it in inadequate doses. Treatment with penicillin greatly reduces the period of infectiousness of syphilis and thus eliminates the sources of infection and the spread of the disease. The use of "depot" penicillin, particularly in the form of crystalline procaine penicillin G in 2 per cent aluminum monostearate, makes possible ambulatory treatment of syphilis, but at the same time it involves the danger of inadequate treatment and the temptation on the part of the patients to regard their infection with syphilis as something of little importance and danger. The cardiolipin-lecithin antigens used at Hasselmann's clinic largely eliminate false positive reactions; at the same time they make possible the quantitative follow-up of the serum titers of the patients during the course of treatment.

Streptomycin in Conservation of Human Milk.—Linneweh stresses that the coli flora in human milk cannot be disregarded. This flora and the souring process was formerly counteracted chiefly by heat sterilization. Since this type of sterilization involves certain disadvantages, the suggestion of Ploetschke to add streptomycin to the milk was accepted. American investigators used streptomycin by mouth in the treatment of children with gastrointestinal disturbances. For more than six months the author studied the use of human milk to which streptomycin had been added. Bacteriologic considerations had determined the addition of 2 mg. (20 units) of streptomycin per hundred cubic centimeters of milk. So far 25 nurslings have been fed with this type of breast milk, comprising either part or all of their feedings, for an average of forty days and for as long as one hundred days. Toxic effects did not result from the streptomycin content of the milk even in the prematurely born infants. From the standpoint of nutritional physiology, milk conserved by the addition of streptomycin is superior to that preserved by heating. Furthermore, the addition of streptomycin to human milk permits simplification of the process of collecting it and increases the supply by reducing spoilage.

Schweizerische medizinische Wochenschrift, Basel

79:465-488 (May 28) 1949. **Partial Index**

Organization of Mass Examinations by Means of Fluorescent Screen Pictures. H. Birkhäuser.—p. 465.
Automatic, Endogenous, Lymphadeno-Bronchogenous Reinfection in Initial Period of Pulmonary Phthisis and Its Typical Sequelae (Concluded). P. Schwartz.—p. 467.
*Congenital Aneurysm of Basal Cerebral Arteries and Polycystic Kidneys. W. Suter.—p. 471.

Congenital Basal Cerebral Aneurysm and Polycystic Kidney.—Suter reports simultaneous occurrence of congenital aneurysm of basal cerebral arteries and polycystic kidney in 10 men and 5 women between the ages of 24 and 56, mentioned in the literature. He reports 2 cases of his own, 1 in a woman aged 56 and 1 in a man aged 36. There seems to be a slight preponderance in the male sex. Both conditions are malformations. Multiple malformations in the same person are considered characteristic of all malformations. Twenty-seven cases of congenital aneurysm of basal cerebral arteries were observed in a series of 5,960 necropsies performed at the pathologic institute of the cantonal hospital in St. Gallen, Switzerland, from 1941 to 1947. In 2 instances in which a congenital aneurysm of the cerebral vessels was demonstrated, there were associated polycystic kidneys. In both of the author's cases death resulted from spontaneous subarachnoid hemorrhage due to rupture of congenital aneurysm of the anterior communicating artery. Bilateral polycystic kidneys were observed in both cases. Both malformations may be placed within the nosologic frame of Bard's syndrome of idiopathic dilatations, or according to Snapper and Formijne they may be interpreted as simultaneous failures in junction of two systems growing toward one another. The prognosis is unfavorable in congenital aneurysm of basal cerebral arteries associated with polycystic kidneys, because of the possibility of a precocious rupture due to nephrogenic hypertension.

Ugeskrift for Laeger, Copenhagen

111:465-496 (April 28) 1949

Extrauterine Pregnancy. Analysis of 109 Cases. A. Christensen.—p. 465.
*Treatment of Abortion with Special Regard to Value of Sulfonamide in Febrile Abortion. E. Østergaard.—p. 496.

Sulfonamide Drugs in Febrile Abortion—During the past four years Østergaard encountered 1,982 cases of abortion; 661 of the patients were febrile. There were 4 deaths. Febrile abortions are complicated secondarily in about one fourth of the cases by inflammation outside of the uterus, which in many cases leaves permanent sequels. The number of these secondary complications can be limited by the introduction of sulfonamide treatment as early as possible in combination with moderately active treatment for conditions related to the abortion. Careful after-examination of abortion patients is important in order that adnexal disorders setting in after the abortion may be discovered and given early treatment.

Book Notices

Clinical Auscultation of the Heart. By Samuel A. Levine, M.D., Clinical Professor of Medicine, Harvard Medical School, Boston, Mass., and W. Proctor Harvey, M.D., Assistant in Medicine, Peter Bent Brigham Hospital, Boston. Cloth. Price, $6.50. Pp. 327, with 286 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1949.

With the introduction in recent years of so many mechanical aids in cardiac diagnosis, a book which sets forth the information that may be gained by simple auscultation is refreshing. Chapters are devoted to heart sounds, cardiac irregularities, cardiac murmurs and miscellaneous auscultatory findings. Along with a description of the acoustic phenomena are heart sound tracings and electrocardiograms. The authors emphasize that these tracings are unnecessary in general practice but are used in the text to illustrate what has been heard and to help visualize the points discussed. They show how by careful auscultation one may be able to make many diagnoses at the bedside without resort to the roentgenogram or the electrocardiogram. In the words of the authors, "When the cost of medical care is necessarily increasing, it is imperative to derive all possible help from such an inexpensive and expedient tool as the stethoscope."

This treatise can be read with profit by every physician who is interested in cardiac diagnosis and is to be especially recommended to the younger physicians to many of whom cardiac auscultation is practically a lost art.

Exploration radiologique de l'appareil urinaire inférieur (vessie, urètre, prostate). Par Bernard Fey, Fernand Stobbaerts, Pierre Truchot et Georges Wolfromm. Avec la collaboration de Léonce Sabadini et al. Paper. Price, 2500 francs. Pp. 291, with 293 illustrations. Masson & Cie, 120 Boulevard Saint-Germain, Paris 6e, 1949.

The authors deal with radiologic examination of the bladder, urethra and prostate in eleven chapters. The first sentence in the opening chapter on cystography stresses a point of ancient vintage, but one in need of constant repetition: "L'examen radiologique de la vessie demande la collaboration de l'Urologue et du Radiologue. . . ."

The first part of the book deals with the authors' technic of cystography, urethrography and combined methods of examination. The second part deals with bladder abnormalities, disturbances of bladder tone, calculi and foreign bodies, inflammatory lesions of the bladder and urethra, dysfunctions of the bladder neck, traumatic lesions of the urethra and bladder, bladder tumors, fistulas and postoperative conditions.

The figures illustrating the traumatic and fistulous lesions of the urethra and bladder are some of the best in the book. The use of double contrast micturition technic for studying the urethra seems to be something of a *tour de force*. The authors do not stress the differential diagnosis between edema of the trigone and intravesical enlargement of the prostate as much as they might. With these minor exceptions, the monograph can be heartily recommended.

Some readers are said to take great pleasure in cutting the pages of a new text; others wish that the printers would attend to this manual chore. With cut pages and a board cover, this monograph would perhaps have greater usage and longer life.

Searchlights on Delinquency: New Psychoanalytic Studies Dedicated to Professor August Aichhorn, on the Occasion of His Seventieth Birthday, July 27, 1948. Managing Editor: K. R. Eissler, M.D., Ph.D. Chairman of the Editorial Board: Paul Federn, M.D. Cloth. Price, $10. Pp. 456. International Universities Press, Inc., 227 W. 13th St., New York 11, 1949.

As an international tribute to August Aichhorn, many of his colleagues prepared this symposium. It represents a psychoanalytic study of the aspects of delinquency, beginning with its causes and general problems and proceeding through social psychology, penology and surveys. The book concludes with a bibliography of Dr. Aichhorn's writings, which are largely concerned with wayward youth. The work will be especially interesting to readers informed on psychoanalysis. The subject is still in an early investigational stage, and readers will find many differences of opinion and points of view.

A Textbook of Practical Nursing. By Kathryn Osmond Brownell, R.N., B.S., Supervisor of School Nursing Service, Connecticut State Board of Education, Hartford, Conn. Third edition. Cloth. Price, $3.75. Pp. 465, with 51 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; 7 Grape St., Shaftesbury Ave., London, W.C.2, 1949.

The author states that this third edition has been revised to include methods and information suggested by the Working Committee on Practical Nurse Training as outlined in the publication "Practical Nursing" (United States Office of Education). Emphasis is placed on the nursing care of the patient, with only brief reference to the household responsibilities commonly associated with the duties of the practical nurse. The statement "There are cases which, due to financial conditions, require housework of the practical nurse in addition to patient care" suggests a changing concept of the function of the practical nurse and appears to indicate that in some respects she is approaching the role of the graduate of a professional school of nursing.

The fact that so much space is devoted to details on the personal habits of the practical nurse, including such matters as bathing and how to spend her free time and her money, suggests that the book is written for a person with a limited background of education, training and experience. The author has attempted to cover all phases of nursing and to use simple terms. In doing so, she has dealt with some subjects so briefly as to sacrifice clarity and in a few instances accuracy. For example, it is stated, "The function of the tonsils is to drain the back of the throat." The chart on communicable diseases reprinted by permission of the New York State Department of Health is the 1943 edition rather than the more recent one of 1948, in which several changes were made to bring it up to date.

The practical nurse is being prepared primarily for giving care to the "subacute, convalescent and chronically ill patient." Since this is so, the book would be much more useful if it included a more detailed discussion on these aspects of illness, particularly the nursing care needed in the chronic diseases. The book is not comprehensive enough to be recommended for general use in the more progressive schools of practical nursing.

Clinical Allergy. By Louis Tuft, M.D., Assistant Professor of Medicine, Temple University School of Medicine, Philadelphia, Pa. Second edition. Cloth. Price, $12. Pp. 690, with 57 illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1949.

The popularity of the first edition of this book was such that it has not been readily available for several years. The author has accomplished a most difficult task by improving an excellent work. Although the number of pages is reduced from 711 to 690, the revision is thorough and includes the most important advances made since the first edition was published in 1937. The book was kept within the original size by the elimination of statistical data, the reduction of the bibliography to general references at the end of the book and by careful editing. The elimination of redundant expressions has led to conciseness and increased clarity.

The organization used in the first edition is maintained with only slight revision because of the added chapters on allergy to fungi and a rearrangement of other material. The book is divided into four sections. The first deals with the fundamental principles of allergy and anaphylaxis. It includes a thorough discussion of the principles of diagnosis and treatment of allergic conditions. Part 1 discusses the various allergic conditions with reference to etiology. Part 3 discusses in seven chapters the various clinical syndromes of allergy with the exception of the dermatoses. In part 4 the allergic dermatoses, allergy in children and allergy in relation to other specialties are discussed. The appendix includes a variety of useful information to the specialist in allergy—laboratory methods in anaphylaxis and in allergy, directions to patients, lists of foods and a variety of miscellaneous information useful primarily to the specialist.

The book was originally aimed specifically for the medical student, the general practitioner and other physicians who are interested in allergy as a peripheral rather than a major field. It is not written as a reference work for the man who is already trained in allergy. Tuft has succeeded in a thorough revision and improvement of a good book.

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Les symptômes de la tuberculose pulmonaire et de ses complications: Clinique, physiologie pathologique, thérapeutique. Par Edouard Rist, médecin honoraire de l'Hôpital Laennec, Paris. Second edition. Paper. Price, 1800 francs. Pp. 983, with 22 illustrations. Masson & Cie, 120 Boulevard Saint-Germain, Paris 6e, 1949.

The first edition of this book was published in 1943. It was thoroughly revised and enlarged by about 400 pages for the 1949 edition. It is an authoritative and extensive presentation (983 pages) of the symptoms of pulmonary tuberculosis and its complications. The book contains fifty chapters grouped into three parts. The first, consisting of eighteen chapters, is devoted to functional symptoms. Cough with reference to cause, treatment and other aspects is discussed in 22 pages. Another chapter is limited to expectorations, including various types of examination of sputum. Four entire chapters deal with blood spitting and frank hemorrhage. Whole chapters are given to voice disturbances and chest pains. Dyspnea and its various aspects are presented in six chapters, consisting of 123 pages. All causes and therapeutic procedures are included.

Part II, on general symptoms, consists of eleven chapters on such subjects as cyanosis, fever, sweat and weight loss. Part III constitutes approximately the last half of the book and is devoted to symptoms of complications and the like. Five chapters deal with gastrointestinal symptoms, including rectal fistulas. Edema and its various causes and treatments, trigeminal nerve disturbances, erythema nodosum, menstrual disorders and pregnancy are a few of the many subjects which are thoroughly discussed.

This book contains a thorough presentation of the symptoms of pulmonary tuberculosis and its complications and the modern methods of controlling them. The author is one of Europe's finest authorities on the subject.

Leisure and Recreation: A Study of Leisure and Recreation in Their Sociological Aspects. By Martin H. Neumeyer, Ph.D., Professor of Sociology, University of Southern California, Los Angeles, and Esther S. Neumeyer, A.M. Second edition. Cloth. Price, $4.50. Pp. 411. A. S. Barnes & Company, 101 5th Ave., New York 3, 1949.

This volume is timely; an inevitable result of our technical progress is more free time for leisure and recreation.

Adequate orientation is provided by chapters on development of leisure and recreation, the recreation movement in this country and other lands, world trends in recreation and conditioning factors in leisure and recreation. Especially helpful is a chapter on leisure and personality, in which reaction of the individual to opportunities offered by increased leisure is discussed. Thought must be given frequently to preparing large segments of our population for leisure if they are to use it and not abuse it. One facet of this problem is treated in a later chapter on recreation and social maladjustment.

The place of public, semipublic and private agencies in development and furtherance of community recreations is described in detail in two chapters. Of considerable value is a chapter on the need for recreation leaders, with emphasis on qualifications and professional training of such leaders. In the final chapter are found important suggestions on methods of research. Inclusion at the end of each chapter of a special section on projects and exercises, as well as lists of selected references, makes the volume a working instrument as well as an important source of general information on this subject.

Gynécologie. Par Albert Netter. Collection médico-chirurgicale à révision annuelle. Directeur général: Pasteur Vallery-Radot. Secrétaire général: Jean Hamburger. Cloth. Loose-leaf. Pp. 762, with 178 illustrations. Ernest Flammarion, 26 rue de Vaugirard, Paris 6e, 1949.

This book is one of a series dealing with medical and surgical subjects. It is in loose leaf form and is to be revised annually as are the other volumes in the series. The book is divided into three parts. The first deals with anatomy and physiology of the genital organs, the second with genital pathology and the third with the mammary gland. The book follows the plan used in most textbooks dealing with gynecology except for the addition of the 107 pages devoted to the breasts. Properly speaking, the breasts are within the province of the gynecologist. Certainly at the time of every bimanual pelvic examination, the breasts should also be carefully examined. There are only 178 illustrations, which is a small number for a textbook of 762 pages. Most of the illustrations are photomicrographs of tissue. At the end of each chapter is a well selected list of references in several languages, and at the end of the book there is a list of authoritative books on gynecology and related subjects. The index is excellent.

The author is one of the leading gynecologists in France, and it is obvious that the book is based on his extensive experience as a practicing gynecologist, and as a pathologist. The advice given for nearly all the disturbances described is in accord with the universal opinion of specialists in gynecology. The language is simple and direct; for those who can read French, the book is highly recommended.

The 1948 Year Book of Pathology and Clinical Pathology. Pathology. Edited by Howard T. Karsner, M.D., Professor of Pathology, Director of the Institute of Pathology, Western Reserve University, Cleveland, Ohio. Assistant Editor: Herbert Z. Lund, M.D., Assistant Professor of Pathology, Western Reserve University. Clinical Pathology. Edited by Arthur Hawley Sanford, M.D., Professor of Clinical Pathology, University of Minnesota (The Mayo Foundation), Rochester, Minn. Cloth. Price, $4.50. Pp. 538, with 127 illustrations. The Year Book Publishers, Inc., 304 S. Dearborn St., Chicago 4, 1949.

The first portion includes abstracts in general pathology, tumors and special pathology by different systems. Of particular interest are the special articles by different authorities on such subjects as the evaluation of exfoliative cytology in tumor diagnosis; pulmonary berylliosis, iron metabolism and the interrelation of vitamin B_{12} and other factors to macrocytic anemia.

The clinical pathology section includes recent contributions to hematology, clinical chemistry, serology, bacteriology, mycology and parasitology. The authors are to be commended for their selection of practical subjects and articles that will be of interest to the clinician as well as the pathologist. Articles contributed by clinical workers are included in which the laboratory findings are discussed not only in relation to diagnosis but also to therapy of the disease.

The Compleat Pediatrician: Practical, Diagnostic, Therapeutic and Preventive Pediatrics for the Use of Medical Students, Internes, General Practitioners, and Pediatricians. By Wilburt C. Davison, M.A., D.Sc., M.D., Professor of Pediatrics, Duke University School of Medicine, Durham, N. C. Sixth edition. Cloth. Price, $4.75. No pagination. Duke University Press, College Station, Durham, N. C., 1949.

This unique book, now its sixth edition, has enjoyed great popularity. Its brevity in spite of its comprehensive coverage of the field together with its unique style puts it in a class by itself.

Although the number of pages have remained the same, 6,500 lines have been changed, and they have been woven into the text sometimes in a most unorthodox style. Space-saving abbreviations are used, and many lines drop off abruptly because of addition or deletion of material. This may bother the new reader of the book until he gets the spirit and purpose of the author. The book was not intended to be read consecutively as a text but was offered as a concise reference book for documentation of practical data useful in everyday practice. The book has been well received by the practitioner and student, and its popularity is well merited.

Odors: Physiology and Control. By Carey P. McCord, the Industrial Health Conservancy Laboratories, Detroit, and William N. Witheridge, Ventilation Consultant, General Motors Corporation, Detroit. Cloth. Price, $6.50. Pp. 405, with illustrations. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1949.

This is the first comprehensive book dealing exclusively with odors and their control. The authors have been interested in the subject for many years. They submit a list of information and material well worth the time of health officers, industrial physicians and others interested in these problems. After careful consideration of the anatomy and physiology of the olfactory system, the third chapter presents methods of detection and measurement of odors. Subsequent chapters are concerned with the influence of odors on health and comfort, odors that appear in the human body in relationship to disease and the value of odors for diagnosis. Then come such subjects as industrial odors, offensive trades and methods for destroying or masking odors. Consideration of the legal aspects of the subject and a comprehensive bibliography concludes this useful work.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

UNDIAGNOSED FEVER

To the Editor:—**A woman aged 62 has had a temperature of 101 to 102 F. in the afternoon and evening for months. The fall of temperature has been associated with profuse sweating. She has general abdominal pain and much pain and aching of the whole body, constipation, loss of appetite and loss of weight. The patient has had an appendectomy, has borne four children and has had no acute illnesses in recent years. The results of physical examination are generally negative; blood cell counts and results of urinalysis are essentially normal. A roentgenogram of the chest shows no sign of active infection; the sedimentation rate is increased. Results of agglutination tests for typhoid, brucellosis and proteus are negative. The result of a skin test with diluted brucellosis vaccine was negative; however, the patient did have an increased amount of sweating and general malaise following the intradermal injection of vaccine. The results of roentgen studies of the gastrointestinal tract were normal. The gallbladder visualized normally.**

M.D., Indiana.

ANSWER.—In the differential diagnosis of an illness of this nature one should consider not only infectious diseases and localized infections but also the possibility of neoplastic diseases. In the further search for infections, repeated cultures of blood, cultures of urine and feces and the examination of the stools for parasites are indicated. The agglutination tests should be repeated if the original ones were done relatively early in the course of the illness. Agglutination tests for the paratyphoid group, tularense and heterophil antibody should be included. In the interpretation of the repeated Brucella agglutination test, it should be remembered that the reaction to the test may become positive for several weeks or months or even longer after a Brucella skin test. Titers as high as 1:320 may be thus engendered, and in these circumstances should not be considered as evidence of brucellosis. If enlarged peripheral lymph nodes are present, a suitable node should be excised for culture, animal inoculation and histologic study. Pyelograms made by the intravenous method may disclose an infectious or a neoplastic process. Examination of the cerebrospinal fluid may be done if other examinations are not fruitful.

Localized infections may produce sepsis with fever and generalized symptoms while showing little or no localizing symptoms. Perinephritic, subdiaphragmatic and liver abscesses are particularly liable to manifest themselves thus. Osteomyelitis may also at times give little localizing symptoms. Sinusitis likewise may produce fever without local symptoms, but suitable roentgen studies will disclose its presence. Full mouth dental roentgenograms should also be made in a search for apical abscesses. In the search for the cause of fever, one must be careful not to overlook tertiary syphilis, extrapulmonary tuberculosis, cirrhosis of the liver, thrombosis and embolism as possible causes.

In the investigation for possible neoplastic diseases gastrointestinal roentgen studies should be supplemented by proctoscopic examination and tests for occult blood in the stools. There should be no hesitation in repeating the roentgen studies if any doubt exists as to their adequacy and completeness or if enough time has elapsed for the further development of a lesion which may have been previously overlooked. Hypernephroma and lymphomas are particularly prone to cause temperature elevation.

The therapeutic trial of aureomycin or other antibiotics before all diagnostic efforts are exhausted is not to be recommended. Such treatment may mask the disease, interfere with diagnostic procedures and render the eventual diagnosis and the subsequent proper treatment more difficult or even impossible.

RHEUMATIC FEVER

To the Editor:—**Can a diagnosis of rheumatic fever be made from any reliable blood test or laboratory findings?**

Paul K. Edwards, M.D., Bloomington, Wis.

ANSWER.—There is no definitive laboratory test for rheumatic fever. Most attacks of rheumatic fever are accompanied by an elevation of the erythrocyte sedimentation rate and an increase in the antistreptolysin O titer of the blood serum. These two blood tests are helpful in the differential diagnosis of rheumatic fever.

TOBACCO AND LONGEVITY

To the Editor:—**What effect, if any, has the use of tobacco on (1) glaucoma, (2) the advance of arteriosclerosis and senility, (3) mental deterioration and (4) longevity.**

Samuel B. Rentsch, M.D., Derby, Conn.

ANSWER.—1. No direct effect of tobacco on glaucoma has been demonstrated. The frequency of glaucoma in women and nonsmokers and the lack of aggravation of symptoms on smoking would argue against the incrimination of tobacco in this respect.

2. Persistent effects of tobacco and of tobacco smoke on the blood vessels have been repeatedly reported, on the basis of experimental studies and clinical observations, but these claims have been vigorously controverted. The immediate effects of tobacco and especially of nicotine on the cardiovascular system are pronounced and definite, but there is a conflict of evidence regarding organic effects following prolonged administration. Some writers have suggested that arteriosclerosis may result secondarily from hypertension aggravated by nicotine, rather than from the direct effect of tobacco on the blood vessels. Statistical studies appear to show a slightly higher incidence of arteriosclerotic conditions among smokers than among nonsmokers, but the effects of associated conditions, such as alcoholism and syphilis, among the smokers cannot be readily excluded.

3. General deterioration of intelligence, especially in juveniles, has been repeatedly ascribed to tobacco smoking, and numerous comparisons of scholastic records and results of mental tests of smokers and nonsmokers have been cited to support this idea. Some of this may represent selection, since the inferior student may more often resort to smoking for consolation or in defiance, but the phenomenon has been extensively confirmed both in colleges and in schools. Whether it would be true of older persons is uncertain, as there are little data regarding the older ages—and many physicians act, at least, as if they doubt it.

4. A shorter life span has been repeatedly alleged to occur in tobacco smokers and has been particularly stressed in an extensive study by Pearl. Some of the excess reported may be ascribed to overindulgence in alcohol and other practices associated with the use of tobacco. Some deaths may be accelerated, if not actually caused, by tobacco smoking, as in Burger's disease and cancer of the lip, but these may cause little change in the total longevity of large groups.

References:

Schrumpf-Pierron, P.: Tobacco and Physical Efficiency: A Digest of Clinical Data, New York, Paul B. Hoeber, Inc., 1927.

Bogen, E.: Tobacco, in Fisher, I., and Emerson, H.: How to Live, ed. 20, New York, Funk and Wagnalls Company, 1938, appendix 11, pp. 253-264.

Pearl, R.: Tobacco Smoking and Longevity, Science **87**: 216 (March 4) 1938.

RETROBULBAR NEURITIS

To the Editor:—**My daughter, age 28, has had retrobulbar neuritis (right) since about Sept. 1, 1948. It followed acute colon bacillus cystitis which cleared up in a few days. She has a baby 7 months old. Vision in the affected eye is still slowly improving. In *The Journal* (138: 1127 [Dec. 17] 1948) there is an abstract of a report by Cerquetelli and Nobili of good results in multiple sclerosis with intravenous histamine injections. Do you think that these injections would be of value in my daughter's case? Information as to treatment and prognosis would be appreciated.**

M.D., Iowa.

ANSWER.—In the absence of more definite information with regard to the cause of the retrobulbar neuritis, the degree of initial loss of vision, the present status of the central vision and fields of vision and the appearance of the optic disk, it is difficult to evaluate the prognosis in this case. If normal vision is to be regained spontaneously after an episode of acute retrobulbar neuritis, recovery will occur in about six weeks after the onset. In some instances, however, gradual improvement will continue for a much longer time. The lapse of nine months, as in this case, would suggest, usually but not necessarily, that the maximum of improvement had been attained.

Because of the tendency to spontaneous improvement and because successful results have been reported mainly when treatment has been instituted early, opinions differ as to the efficacy of any therapy for retrobulbar neuritis or other episodes in multiple sclerosis. A number of ophthalmologists think that vasodilatation promotes recovery in cases of retrobulbar neuritis and improves the visual end results. In competent hands, the intravenous administration of histamine has seemed to be a safe and satisfactory method of producing vasodilatation in the optic nerve and retina. Since it is impossible to tell, on the basis of elapsed time alone, when actual atrophy of the optic nerve fibers has occurred, it seems justifiable to try a course of intravenous histamine therapy at any stage in the course of retrobulbar neuritis, provided atrophy is not visible ophthalmoscopically in the optic disk.

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BLACK TONGUE

To the Editor:—Is there any recent treatment for "lingua villosa nigra?" The patient in question has dissimilar tooth fillings and a vulcanite plate. He had coronary disease a year and a half ago; otherwise he appears to be in good health.

M.D., Massachusetts.

ANSWER.—There is no new or specific treatment for "lingua villosa nigra," called also black tongue, hairy tongue and lingua filaceosa chromatica. The lesion is a hypertrophic elongation of the filiform papillae, similar to keratosis of the skin. The cause is uncertain, and the various yeasts and fungi which may be found are secondary. The dental condition is probably also unrelated.

The patient should be told that the condition is harmless, that therapy is aimed at clearing away the offending growth and that it may recur. The area may be scraped or brushed every few days; 1 per cent aqueous acriviolet (a mixture of acriflavine and methylrosaniline chloride), methylrosaniline chloride or hydrogen peroxide (3 per cent) may be applied recurrently, and the use of strong sodium chloride solution or sodium perborate (and other boron) mouth washes should be banned, since they are considered by some to be causative.

A new cause for black tongue has been described, "Black Hairy Tongue from Penicillin" (Wolfson, S. A., J. A. M. A. **15:** 1206 [Aug. 13] 1949), with a review of the foreign literature. Since the connection would be obvious from a history, and since the lesion results from local use of the drug, withdrawal should clear the condition.

DEPOSITS ABOUT SHOULDER JOINTS

To the Editor:—I have several patients with calcific deposits in soft tissue about shoulder joints and hypertrophic osteoarthritis of bodies of the cervical part of the spine. The ages of the patients range from 49 to 57 years. No foci of infection have been discovered, and the results of physical examination are otherwise normal. The patients' complaints are pain and slight limitation of motion of the involved regions.

M.D., New York.

ANSWER.—In the literature on the two subjects, no emphasis has been placed on the coexistence of osteoarthritis of the cervical vertebrae and calcific tendinitis in the shoulder and there is no mention of the shoulder condition's being secondary to the cervical pathologic condition. However, Fletcher points out in the *British Journal of Physical Medicine* (**8:**45-51, 1945) that in osteoarthritis of the cervical part of the spine there is a common pattern of pain radiating over the top of the shoulder down to about the level of the insertion of the deltoid. Meyerding and Ivins present a discussion of the differential diagnosis of the painful stiff shoulder in the *Transactions of the Western Surgical Association* (**55:**155-166, 1947).

The roentgen observations in the cases described are the clues to the underlying pathologic conditions. Since complaint is made of pain and limitation of motion in the "involved regions," both the shoulder and neck should be treated by first heating with infra-red radiation or short wave diathermy for thirty minutes followed by deep sedative type massage to both and then assistive and, finally, active exercises to regain the normal range of motion in each. This routine of treatment can be carried out once or twice daily. After each treatment selected patients should have mild cervical traction with the Sayre sling according to the procedure described by McFarland and Krusen (*Arch. Phys. Therapy* **24:**263-269 [May] 1943).

TRANSFUSION OF LEUKEMIC BLOOD

To the Editor:—Should blood from a person with chronic lymphatic leukemia be used for transfusion? Is there a possibility of transmission of leukemia this way? Has anyone reported treatment of leukemia by transfusion with blood from donors who were first "immunized" with leukemic blood? Would massive transfusions of this "antileukemic" blood with simultaneous removal of blood from the recipient be worth trying if the assumption that an antileukemic factor can be produced in donors is correct?

G. D. Gregory, M.D., Chicago Heights, Ill.

ANSWER.—There are several reports relative to the transfusion of leukemic blood. In each instance there was no evidence of transmission of leukemia. Thiersch in over 50 cases did not observe the development of leukemia in human subjects after subcutaneous injection of leukemic blood, lymph node or splenic tissue. It has been possible to transmit leukemia as well as certain cancers in an experimental animal such as the mouse only if the animal is of selected, inbred strain.

There is no evidence that any anti-human-leukemia factor has been produced by immunization technics, as far as known.

Bessis treated 38 patients with acute leukemia by massive blood replacement transfusions and obtained improvement in 30. He believes this to be caused by an antileukemic factor present in normal blood. However, this work needs further corroboration. Dr. N. Rosenthal has tried this replacement transfusion technic on a large number of patients with acute leukemia, and he finds a lack of evidence that the course of the disease is significantly influenced.

TEST FOR PHEOCHROMOCYTOMA

To the Editor:—A married woman aged 52 complains of paroxysms of rapid and irregular heart beating with elevated blood pressure and dyspnea. Between the attacks her blood pressure subsides to 160 systolic and 90 diastolic. During and after the attacks her pressure ranges from 190 systolic and 100 diastolic to 200 systolic and 110 diastolic. The attacks come at irregular intervals of about one week. One of them occurred one hour after she took her first and only dose of priscoline® (2-benzyl-4,5-imidazoline hydrochloride), 25 mg. orally. It was the severest up to the present time and occurred on the evening of one of her best days. The electrocardiographic tracing shows left axis deviation with moderate myocardial degeneration. Retrograde pyelograms show grade 1 hydronephrosis of the right kidney. The right kidney is rotated. When the patient is placed in the erect position, neither kidney descends more than a few centimeters. No soft tissue mass is made out at the kidney area. Is there any test short of exploration by which pheochromocytoma may be excluded as a cause of her attacks of paroxysmal tachycardia.

Paul R. Farrington, M.D., Boulder, Colo.

ANSWER.—It does not seem likely from the history given that the patient referred to has pheochromocytoma. As a rule the increase in blood pressure paroxysmally is greater than that recorded here and not necessarily dependent on arrhythmia, if the rapid irregular heart beating was auricular fibrillation. There can be a pressor effect from paroxysmal tachycardia itself, and, since there was slight hypertension to start with, this patient would likely be more prone to such hyperreaction. It is true that with pheochromocytoma there tends to be tachycardia as well as increased blood pressure, but that is sinoauricular and not paroxysmal and often there are other symptoms, too, such as increased perspiration.

The recent introduction of piperidylmethyl benzodioxane (933F), which specifically reduces the blood pressure in cases of pheochromocytoma, has been found reliable and preferable to the test with histamine; it may be used helpfully in the patient referred to, to exclude pheochromocytoma. The article by Goldenberg, Snyder and Aranow refers to this test (J. A. M. A. **135:**971-976 [Dec. 13] 1947).

SUNBURN

To the Editor:—After viewing the amount of sunburned skin during a visit to a beach, my curiosity was aroused as to the possible after-effects of sunburn and prolonged deep tanning. Have any studies been made regarding possible renal damage as a result of such exposures? I heard a dermatologist express concern about the possible effect of such tanning on women's skin in connection with the later appearance of epitheliomas. are there any data about sunburn or tanning as an etiologic factor in epitheliomas?

M.D., Washington, D. C.

ANSWER.—Renal damage per se does not occur from exposure to sunlight, although it may occur as part of the syndrome of heatstroke.

Practically all textbooks mention exposure to strong sunlight as one of the factors that may lead to the development of skin cancers. Such lesions are much more prevalent in sunny countries, on sun-exposed areas of the face and in those engaged in outdoor occupations. Sunlight, however, does not usually constitute a hazard to the person who courts the sun at intervals only, such as on weekends and during vacation periods during the summer months. There have been many studies made of the effect of sunlight on the skin. References to them are given in most full textbooks on dermatology.

HYPOCHLOREMIA

To the Editor:—In the May 14 issue of THE JOURNAL, in the Queries and Minor Notes, there is a reply to a letter on the subject of hypochloremia. It seems to me incomplete, as it fails to recognize that the patient described had been on a salt-poor diet previous to the paracentesis in which 2,000 cc. of the fluid was removed and the wound allowed to drain. My associates and I have observed the syndrome described in just these circumstances and have found a decrease of the blood chlorides. The relief obtained from relatively small amounts of isotonic sodium chloride solution given intravenously is dramatic. The explanation seems to be that when there is prolonged use of a salt-poor diet the electrolyte reserves of the body are depleted and cannot stand the additional loss entailed by the removal of ascitic fluid. The same syndrome has been produced experimentally in patients by continued removal of gastric juice and subsequent hypochloremia (Kirsner, J. B.; Palmer, W. L., and Knowlton, K.: Studies on Experimental and Clinical Hypochloremia in Man, J. Clin. Investigation **22:** 95, 1943):

Walter L. Palmer, M.D., Professor of Medicine, University of Chicago.